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Table of Contents

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ORIGINAL ARTICLES— PAGE.

The Bancroft Memorial Lecture: "Medical Research in Australia", by W. J. PENFOLD, M.B., Ch.M., B.Hy., M.R.C.S. 707

"Intrabiliary Rupture of Hydatid Cysts of the Liver: Report of Five Cases", by ARTHUR L. CARRODUS, M.B., B.S. 714

REPORTS OF CASES—

"Polycythæmia", by J. F. CHAMBERS, M.D., M.R.C.P. 724

REVIEWS—

Materia Medica and Therapeutics 726

Forensic Medicine 726

LEADING ARTICLES—

Doctors' Hobbies 727

CURRENT COMMENT—

Anæmia in the Chronic Rheumatic Diseases .. 728

Icterus Neonatorum 728

Abscess of the Lung and Dental Operations 729

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.

Pædiatrics 730

Orthopædic Surgery 730

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 732

Nominations and Elections 736

CORRESPONDENCE—

X Ray Treatment of Acne 736

An Intermediate Ophthalmic Service 737

Treatment by Olive Oil Emulsion 737

BOOKS RECEIVED 738

DIARY FOR THE MONTH 738

MEDICAL APPOINTMENTS VACANT, ETC. 738

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 738

EDITORIAL NOTICES 738

The Bancroft Memorial Lecture.

MEDICAL RESEARCH IN AUSTRALIA.[1]

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FOR the evening dedicated by the Queensland medical profession to the memory of Joseph Bancroft, probably no more fitting subject for discourse could be chosen than medical research in Australia. It is an extensive subject, and I would ask you to permit me to restrict my consideration of it to happenings of the last twenty years. This represents the length of my sojourn amongst you, so that the aspects of research with which I deal will have not infrequently an almost personal character. As a Cumbrian, I greatly appreciate your invitation to assist in keeping before us the value of the work of a Lancastrian who found in Australia a home and congenial work.

In 1916 in London I was appointed to the Directorship of the Commonwealth Serum Laboratories. On appointment, my colleague, Dr. Ledingham, now Director of the Lister Institute, advised me against accepting it, and gave as one of his reasons: "You will always be at least six weeks behind the times", to which I replied: "Perhaps the time is coming, Ledingham, when you will be six weeks behind the times." This attitude of expectancy towards the publication of new results illustrates the pleasure felt by a devoted scientist to advances in his subject. My reply was no less sincere, but as I look back I am afraid it was less justified.

Let us consider the factors that make for success in the pursuit of scientific research.

First, we require suitable personalities to undertake the work. I believe that in such personalities

[1] Delivered at a meeting of the Queensland Branch of the British Medical Association on May 31, 1935.

Australia is rich. The type should have, first, an insatiable curiosity; secondly, energy and devotion in the work; and thirdly, a consciousness of high purpose, and it should find pleasure in evolving that body of harmonious thought in the subject studied that we poor humans call truth. I have said I have found Australia to be rich in such personalities. How can I think otherwise when Willis, Tiegs, Trumble, Cox, Travers, Kaye and many others have come to work at the Baker Institute without hope of fee or material reward.

The second condition precedent to success is reasonable financial support of the work. Has Australia been generous in the material support it has granted to medical research? I would answer that it has. When we consider the legacy left by the war and the recent depression, we cannot but be appreciative of the earnestness of the public. If we take the subject of research into the causes and treatment of cancer, we are astounded at the money devoted to the purpose. In Sydney, for example, a generous public subscribed about £137,000 for this work. The Commonwealth set aside £100,000 for the purchase of radium to see what it could do to control the disease and alleviate the suffering of the cancer patients. The University of Melbourne also controls a fund for cancer research endowed by the late Sir John Grice, Mr. Fletcher and others. Many smaller sums have been devoted to this subject, so that it cannot be said that the material support of cancer research in Australia has been small.

When we consider the provision made for the study of tropical disease, we find that Australia has had two centres for the purpose: one at Townsville, financed initially by the Commonwealth and Queensland Governments and the Australian universities. It started in 1909. In 1911 it was transferred to the Commonwealth Department of External Affairs, and in 1921 was placed under the Health Department. In 1925–1926 its expenses were £9,363, and in 1926–1927, £9,278, so that it was liberally financed until it was discovered that it was in the wrong place. The Sydney University School of Public Health and Tropical Hygiene was then mooted, and an arrangement entered into between the Commonwealth and the University whereby the new school was to be built at a cost of £30,000 by the Commonwealth Government, and to be financed by the same Government to the extent of £9,000 *per annum* for ten to twenty-five years.

Both of these centres were to be schools of tropical medicine and centres for research in the subject. The Sydney school was also to embrace public health generally in its purview. There are not many countries of Australia's population which can boast of having had two schools of tropical medicine.

In respect of the laboratory side of general medicine, Australia has been equally lavish in the provision of centres for research. Several of the States have provided themselves with laboratories for public health and investigational purposes, and in the last twenty years in these laboratories Cleland, Bull, McGillivray and many others have obtained new and important results.

The Commonwealth has created the Commonwealth Serum Laboratories at Royal Park, and no less than nine subsidiary laboratories throughout Australia. These are all spacious and well equipped, and have adequately paid staffs. The central laboratory has commercial functions in producing biological preparations, but I believe it has also the finest material facilities for experimental medical research found in the Commonwealth today. The subsidiary laboratories carry out routine tests in clinical pathology and public health and also research. The research output of these Commonwealth laboratories has been surprisingly small when one considers their number, their elaborate staffs and their financial resources. I am not aware that any one of them has succeeded in grouping round it a body of voluntary workers of conspicuous devotion and capacity.

Of the privately endowed laboratories for medical research mention must be made of the Walter and Eliza Hall and the Baker Institutes, both in Melbourne, the laboratory built by the Rockefeller Foundation in Sydney at a cost of £100,000, the Kanematsu Institute at the Sydney Hospital, and the Medical Research Institute of the Royal North Shore Hospital of Sydney. Usually in these laboratories routine duties are carried out as well as research. In three of the States the university departments of pathology, physiology and anatomy also provide excellent facilities for laboratory research.

Clinical research, similarly, has not been neglected. The sum of £10,000 was devoted by the Edward Wilson (of *The Argus*) Trust to the investigation of the cause and control of maternal mortality, and the princely Bosch endowments of professorships in medicine, surgery and bacteriology, at a cost of £250,000, should guarantee, as far as material resources can, fruitful schools of clinical research in the University of Sydney.

Research in Clinical Medicine.

Professor C. J. Lambie, of the University of Sydney, in THE MEDICAL JOURNAL OF AUSTRALIA, January 31, 1931, page 125, has supplied us with a sketch of a research staff; and with the princely Bosch endowments I sincerely hope that it may be possible for him to realize his desires in this matter. He would have attached to the Department of the Professor of Medicine, first, a number of part-time qualified men desiring to be consultants ultimately; secondly, whole-time research workers representing the fundamental sciences, men, however, who would not be suitable to become professors of medicine. In this regard he gives a very unfavourable judgement on physiologists in the guise of clinicians, a judgement to which I cannot subscribe as I have been intimately associated with a number of physiologists who became first-class clinicians, from Professor Hugh MacLean downwards. Thirdly, he would like academic physicians as whole-time or almost whole-time assistants to the Professor of Medicine. The academic physician, according to Lambie, should be a man fitted by training in the fundamental sciences and by experiences of the practical physician to appreciate the aims, methods and spirit of both

practice and research, to inspire and promote investigation both at the bedside and in the laboratory, to undertake the care of patients, and to teach the principles of the institutes of medicine. It seems to me that the academic physician pictured must combine in his own person all the qualities that we usually associate with a team of workers, and while this self-culture may be the goal of some academic physicians, I question if it is the best ideal that they could pursue. If we could get all the qualities of Lambie's academic physician in a team of two or three workers who could cooperate in harmony, I cannot but think it would be a more effective method as far as research is concerned. I am sure that the Australian medical profession is looking forward to great things from Professor Lambie and his staff, when he can elaborate it.

The Development of a Medical Research Institute in Australia.

Several institutes in Australia have relatively small endowments, and it becomes a question of policy as to what should be done with the small endowments they possess. With the profession interested in these institutes I should like to discuss the policy of the Baker Institute.

After providing for the routine work, the first thing to be done is to provide the facilities for good medical research work. Those facilities include, firstly, a good animal house well stocked with the species of animals usually used in medical research; secondly, a number of technical assistants highly trained in the techniques of bacteriology, physiology, biochemistry and pathology, these assistants to be ready to assist clinicians who have research leanings but are without technical training. Specialist physicians with an intimate knowledge of the literature of their subject can then associate themselves with the institute and have the difficult technical portions of the work carried out for them, and their responsibility will be to keep up to date in the literature, to interpret experimental results correctly, to project new experiments as the subject opens up and to require such control of the work as may be necessary to establish the truth of the new results obtained, the results to be published in joint papers.

A good librarian, with a knowledge of languages, must be available in the institute to make translations from those languages which are unknown to the physicians and to give every assistance in the literary research necessary to prevent the part-time investigators being ignorant of something which is essential in their work. Then someone should be available in the institute accustomed to the methods of research, having a reasonable knowledge of modern languages, a good imagination and critical faculty, and a real joy in helping others to present their work in such a way that it is lucid and worthy.

To do the preparatory work in building up such an institute requires about two years, but after that it should become a place of resort of earnest clinicians, whether medical or surgical, and should be of the utmost value to them in the investigation of their clinical problems. The senior members of the technical staff must be well paid to keep them attached to the institute and to train the younger technicians. The most difficult technique carried out in the Baker Institute can be perfectly well undertaken by enthusiastic youngsters of no more than twenty or twenty-two years of age. This foundation for the work of a research institute involves only a moderate amount of expense, but it will attract so many honorary workers that the output of the institute will be greatly increased and the cost of the work will be ultimately reduced.

Medical Research Partnerships.

Modern medicine is so complicated that the clinician who would carry out medical research without a biochemist, bacteriologist and/or pathologist is frequently in an extremely weak position. A laboratory physician likewise requires the help of similar officers, and it is well that we should have a clear understanding of the unwritten deed of partnership existing between them. The clinician formerly looked on these accessory officers as constituting a service which supplied him with essential particulars about the case or disease being investigated. The results of their work were his personal property because the cases were his cases. The laboratory physician similarly was liable to appropriate the results of the work of his associates because he had outlined the scheme of work and supervised its execution. It is in the highest degree advisable that these attitudes should be revised. Let the experts of the ancillary sciences be treated as colleagues and partners in the work, as they deserve to be, and I am sure our progress would be infinitely greater than it has been. The old idea that collaboration with workers who had not medical degrees was unprofessional must go, and we would be wise to have a register of medical biochemists and medical bacteriologists who may work with us on a footing of equality. These officers should be properly qualified to do medical work in their subjects, and should be registered in suitable categories in a medical register.

Having admitted these workers to partnership status, the working of the partnership may be found to present some difficulties, but difficulties which should not be insuperable with those who are united in the common ideal of furthering the progress of medicine. Where such a partnership exists, the results of the work should be published as joint papers. It is unbecoming of one of the partners publicly to claim personal rights in a portion of the research. Suppose, for example, one worker draws up a plan of a research after much reading and thought, and another worker carries out portion of that research, and in carrying it out observes something of interest, is the new fact observed to be attributed to the partnership or is it to be attributed to the partner who observed it? I suggest that it is due to both of them, the one who set the stage for the observation and the one who made it. A partnership on any other basis can only result in undignified wrangling. Workers who desire to have exclusive proprietary rights in the results of research should not enter a partnership.

The Association of the Old and the Young.—We are not uncommonly treated to dissertations on the antagonism between age and youth, but I have been fortunate in my experience in research in this matter. I have been associated with younger colleagues who have been physically much more vigorous than I, and while I could contribute a plan of research and secure good control of the work and vary the plan of it according to a fairly wide experience, the actual doing of the work has to a large extent been left to them. Such a basis of partnership I find to be extremely satisfactory, but I work it with this proviso, that as soon as the young worker feels that his acquaintance with the subject is such as to fit him for independent work, he will express the desire for such freedom, when our partnership will be amiably dissolved and our friendship will be unimpaired.

As long as the partnership subsists, however, there should be no claiming of personal as opposed to joint results. It is difficult for the clinician to adopt this standard. It entails to a certain extent a diminution of his proprietary rights in the clinical material, and to that extent a diminution perhaps of his personal importance, but it is to the advantage of the patient and to the advantage of the progress of our science.

In reference to "age", I heard Osler speak in 1914 at the Aberdeen meeting of the British Medical Association dinner. Of Ogston, the president, surgeon and bacteriologist, he said: "He has taught us how to mature." One of the Mayo brothers, recently the recipient of birthday congratulations, showed how greatly he had valued the opportunity of helping his younger colleagues who were climbing the ladder of surgical success, which he was descending. So much was this the case that the descent was as agreeable as the ascent. These ideas express the relationship which should subsist between youth and age in our research.

Patronage of Research.

The patronage of research is in the hands either of trustees of private endowments or of governments and their officials. In the Baker Institute we had our endowments almost entirely from private sources, namely, from Mr. Baker and other members of his family, and so interested was Mr. Baker himself, personally, in the work, that he acted as Chairman of the Trust and attended our meetings once a fortnight without ever missing a meeting until his death. During that early period of our work he would allow nothing to interrupt us in the development of the institute. He was extremely generous and sympathetic, and anticipated, when he retired from business, to come and work with us. After his death unfortunately a period of depression came upon our country. It was then that we realized what a friend he was to research and to the research worker. Such love of research as he had was not widespread amongst the public. The testing time has not on the whole been harmful, and has given the staff and the trustees an opportunity of showing their devotion. As a result of Mr. Baker's provision his interests in Kodak are now paying for the upkeep of practically the whole institute, and ultimately one-third of the Kodak Company's dividend will be devoted to the support of the institute and other charitable objects.

The Government Patronage of Research.

In England, just before the beginning of the War, the Government determined to conduct medical research, and the question arose as to whether this medical research should be conducted under the Ministry of Health or not. The feeling of Parliament was that it should be, but the Ministry of Health, I was informed by Sir Morley Fletcher himself, took up the attitude that it had not been constituted for research purposes, and that there were many men in the country who had devoted their lives to research from whom the Government could select a personnel which would make an effective medical research authority. This unselfish and enlightened attitude of the Ministry of Health has been of great value to the progress of medical research in England. Men like Murray, the discoverer of the thyreoid treatment of goitre, Hopkins, the discoverer of the growth vitamin in milk, Leishmann, the discoverer of the cause of kala-azar, and many other workers of similar standing have been on the Council. They were chosen because of their individual records in research. They had been successful and devoted research workers. They did not represent corporations. The majority of the Council has always been composed of scientists of this calibre, but in addition Parliament has been represented by two or three persons to see that the funds of the Council were spent for medical research purposes as Parliament desired, and to be able to deal with any questions affecting the Council that might arise in Parliament. The presidency of the Council has been held by outstanding men like Lord Moulton, himself a great scientist, and Earl Balfour, a detached philosophic thinker.

This Council controlled the disbursement of about £140,000 annually. A certain amount of this, approximately £50,000, was and still is devoted to work in its own central laboratory. The rest is disbursed chiefly to workers in other laboratories, where the Council feels assured successful results may be expected. The Council has allowed the greatest freedom to the workers, but the junior workers naturally have as a rule been working under senior men with some experience. I have never heard any serious adverse criticism of the Council, and the results that it has sponsored have been exceptionally reliable and authoritative.

Government medical research in Australia for some reason or other seems to attract the Federal Health Department, and in spite of the failure of the Townsville Institute for Research when it was under departmental control, still another research institute in Sydney has been put under similar control. It was suggested by many witnesses before the Standing Committee on Public Works that the school of teaching and research in public health and tropical medicine in Sydney should be placed under the control of a Medical Research Council. This was

opposed by Dr. Cumpston, the Director-General of Health, who stated:

> It would be quite impracticable to hand the control of the school to the Medical Research Council. This is to be a teaching school and the Medical Research Council would not be composed of persons having any administrative relationships to public health. It would be like handing over a military training school to a body of university professors. The Medical Research Council in Great Britain has nothing to do with the School of Health and Tropical Medicine. I should like to state my conviction that such an arrangement would entirely destroy the whole value of the establishment.

Such animosity to a Medical Research Council came as an eye-opener to myself. The Council's record in Great Britain had been so wonderful that when I talked to the acknowledged leaders of our profession in England in 1926 they had nothing but praise for it. This statement of Dr. Cumpston relative to the Medical Research Council in England needs correction. He said the Medical Research Council in Great Britain has nothing to do with the School of Health and Tropical Medicine. On looking up the constitution of the Court of Governors of the London School of Hygiene and Tropical Medicine, I find the Medical Research Council was represented on the Court, in the year 1924, for example, by Sir Frederick Andrewes, a member of the Council. Sir Morley Fletcher, its secretary, likewise had a seat on it. Sir Morley Fletcher was also on the small Board of Management, so that Dr. Cumpston's evidence to the Parliamentary Standing Committee on Public Works was misleading in this matter. Further, the committee advising Sir Alfred Mond, the Minister of Health, as to the site and planning of the School of Hygiene had Sir Morley Fletcher (secretary), Sir William Leishmann (member), and Sir Henry Dale (an employee of the Research Council) on it, out of a committee of nine members, so that the Medical Research Council was represented.

How the value of a school of public health and tropical disease dealing with teaching and research in these subjects could be entirely destroyed by control by a Medical Research Council constituted on the English model is past my comprehension. If we had such a system of management in Australia, it seems to me that the school would provide a very gratifying control of the Health Department's efforts as a research authority.

The Health Commission of 1925–1926 in Australia recommended a certain type of Medical Research Council. I take exception to it because the members are not nominated on account of their personal history as research workers. The first member to be proposed is a representative of the Government, who should be a business or financial authority, to be appointed by the Governor-General in Council. That seems a reasonable provision. The next member suggested is the Director-General of Health, who should be a member *ex officio* so that presumably he would be the only member of the Council who would be continuously in office. Then representatives of the universities are suggested, nominated by their faculties of medicine. This representation of corporations has not been adopted by Great Britain, and is very unwise. If these faculties have outstanding research men, it would be much better for them to be selected on their personal merits. If the faculties are asked to nominate members, some very unsuitable men may very well be found on the Council.

The Federal Committee of the British Medical Association, it is suggested, should send three representatives, one of whom should be or have been engaged in research. Now the Federal Committee of the British Medical Association is not a research body. It is composed very largely of those interested in medical politics, which are doubtless extremely important from the standpoint of the profession, but such politics are no training ground for the patrons of research. The Health Commission is to be congratulated that it insisted that "one" of these members nominated by the Federal Committee should have had experience in research. A scientist, further, was to be nominated by the National Research Council. It does not seem to be necessary apparently that any of the members should have had extensive experience of medical research, although we may assume that not less than two would have had experience of research in some form or other. This scheme of representing corporations and making the members the patrons of medical research in Australia leaves much to be desired. I think we would be wise in selecting the personnel of our Medical Research Council to restrict our selection of the scientific members entirely to those who have got outstanding records of personal medical research work. If we have such men administering the research grant and selecting the personnel, we can be assured that the research workers will have no other anxiety than to get good results from their work. The duties of a Medical Research Council, if they are going to be of value to the State, must include the absolute control of the grant.

The next thing to be insisted upon is that the workers of the Medical Research Council must not work under Public Service regulations. They do not do so in Great Britain, and there are grave reasons why they should not do so here.

Coordination and Overlapping.

In reference to research no word inspires me with greater dread than "coordination". Some authorities believe apparently that there is a real risk of two persons discovering, let us say, a cure for cancer at the same time. If A and B are engaged in the search it is really necessary, according to these coordinating authorities, that A and B should communicate their proposals and *interim* results to C, who will coordinate them and prevent A and B overlapping in their further work. The idea of the coordinator C seems to be that he should be a higher controlling official who must incidentally have a higher salary than the research workers. It is extremely frequent that two persons attack the same subject in the same way, but nevertheless they do not get the same results. One may observe what the other misses, and so on. Coordination of research work should be direct. A and B should address each other, but both should be first-class minds operating in freedom, and if both feel they should be doing the same work, then I would say

let them do it. A thoughtful, capable, independent worker should not be put off his research by any coordinator. The coordinator soon finds that such a worker will not be put off, and then he is driven to seek out second-class minds who are coordinable, and so a team is produced of ineffective nonentities. The only persons who can effectively control research workers are men who have themselves been devoted and successful researchers, men who will inspire respect and affection in their juniors. Their control will be of the nature of suggestion and service and will have a spiritual quality undreamt of by coordinating officialdom. The Townsville Research Institute was controlled from Melbourne by the Director-General of Health, who directed the Director of Tropical Hygiene at Brisbane, who directed the Director of the Institute (who was usually in the Mandated Territory), so that there had to be an acting-director on the spot who actually did direct the institute. This chain of directors could effectually block progress. The research workers under the acting-director occupied the fifth rung of the ladder downwards. Such a scheme of coordination is unduly expensive and cannot work well.

Public Health Experiments in Australia.

Joseph Bancroft was a member of the Board of Health of Queensland, and must have had wide sympathies with public health endeavour. It is peculiarly fitting, therefore, that we look at some of our Australian experiments in public health. It is one thing to say that anthrax serum can cure anthrax; it is another thing to arrest the national mortality from it. Similarly, toxin-antitoxin may be an effective prophylactic of diphtheria, but it may not be easy to prevent diphtheria nationally.

The public health experiments that I should like to consider will deal with the control of anthrax, bovine tuberculosis in children and bovines, diabetes, snake bite, tapeworm and beef measles, and diphtheria.

Anthrax.

It was shown by Sclavo, of Siena, that a good curative serum could be made in the ass against anthrax. Further work was done in Budapest, in the United States of America and elsewhere, which showed that a similarly effective serum could be made in the horse. In these circumstances it seemed unreasonable that we should have deaths from anthrax in Australia. The deaths that we had depended mainly on the inoculation of open wounds by the infected shaving brush, so that the disease usually started in the shaving area. The control of this disease by administrative action was not effective. Arrangements with foreign governments and the seizure of batches of brushes suspected of being infective were unavailing in the control of the disease. While I was at the Commonwealth Serum Laboratories, therefore, I had three horses so immunized against anthrax that they could withstand a very large dose of the virulent organisms. The serum from the third horse was a most excellent neutralizer in guinea-pigs of virulent anthrax culture and much superior to serum that we had imported from abroad for control test purposes. This serum was therefore distributed amongst all the quarantine depôts and various hospitals in such a way that wherever a case of anthrax occurred in Australia the serum was accessible. The effect on the mortality from anthrax in Australia is shown in Table I. (Figures from Dr. Rowan, Victorian Health Department.)

TABLE I.

Anthrax Mortality in Australia.

Year.	Before Anthrax Serum was Available.	Year.	After Anthrax Serum was Available.
1914	2	1924	0
1915	1	1925	1
1916	5	1926	0
1917	2	1927	0
1918	1	1928	1
1919	4	1929	1
1920	3	1930	0
1921	6	1931	1
1922	3	1932	1
1923	3	1933	0
Total for 10 years	30	Total for 10 years ..	5

During the four years immediately preceding the issue of this serum fifteen deaths had occurred from anthrax in Australia, while in the succeeding four years after the issue of the serum only one death occurred. This experiment was done without any government appropriation. It was done out of the earnings of the Commonwealth Serum Laboratories as a piece of the routine work, and its success depended on issuing a good serum and having it properly distributed. Availability of supplies is a very important factor in public health work.

Bovine Tuberculosis.

The control of bovine tuberculosis in children and bovines has been attempted in Victoria with very poor results. Partial compensation is paid to the dairy farmer who will have his herd tested by means of tuberculin for the detection of tuberculosis and who has the reacting animals slaughtered. This work has been carried on now in Victoria for several years, but in an unsystematic manner, and herds have been freed of tuberculosis simply to be reinfected through the lack of effective supervision of our butter factories and of the dairy industry generally. In this regard I would refer you to the paper by Rudduck in THE MEDICAL JOURNAL OF AUSTRALIA of April 27, 1935. At a relatively small cost and with a thoroughly well thought out and controlled scheme, it would be possible to free of tuberculosis the dairy herds supplying Melbourne children with milk. With a proper system of quarantine and butter factory supervision and the use of the tuberculin test, it should be possible to keep our dairy herds healthy. Now that the Country Party has obtained power in Victoria, they should make it a point of honour to deal with this question. Bovine tuberculosis is still very prevalent in children, as indicated by Webster's figures given in THE MEDICAL JOURNAL OF AUSTRALIA of March 16, 1935.

Diabetes.

Mr. Thomas Baker, the founder of the Baker Institute, sponsored a scheme for reducing the diabetic mortality in the State of Victoria. The scheme consisted in an educational campaign of doctors on the one hand and of patients on the other and had other features, some of which have not yet been successfully brought into operation. The scheme in recent years has been in the hands of Dr. Ewen Downie, head of the Diabetic Clinic of the Alfred Hospital; and his analysis of Victorian diabetic mortality shows that there has been a great saving of life in Victoria under fifty years of age, even though there has been no marked change in the general death rate from diabetes. The diabetic deaths have been occurring chiefly amongst old women. His comparison of the position of diabetic mortality in New South Wales as against Victoria is definitely in favour of Victoria.

Snake Bite.

Since 1927 the Walter and Eliza Hall Institute and the Commonwealth Serum Laboratories have worked on the subject of the reduction of the mortality in Australia from snake bite. This costly research work has been long continued, and has supplied many interesting new facts on the subject, but has also revealed the extreme difficulty of favourably influencing the public health situation. The recent mortality from snake bite in Australia, apart from that of the year 1933, has not been very different from what it was some years ago. (See Table II.) (Figures from Dr. Rowan of the Victorian Health Department.)

TABLE II.

Deaths from Snake Bite in Australia.

Year.	Number of Deaths.
1914 to 1927	During these years the deaths were grouped under "Venomous Bites", but it is estimated that there was an average of 10 deaths *per annum* from snake bite, approximately.
1928	10
1929	9
1930	12
1931	10
1932	13
1933	6

Tapeworm and Beef Measles.

The Melbourne and Metropolitan Board of Works has employed the Baker Institute to investigate the occurrence of beef measles, with a view to its control. I have the Board's permission to mention briefly some of our results. This work has been carried on for about sixteen months by the institute, and has resulted in a substantial increase in our knowledge of the general problem. An effort has been made to rid the human carriers of tapeworm and so arrest the discharge of tapeworm eggs. Moreover, field experiments at the Board's farm have resulted in disclosing fresh possibilities of control by the production of an active immunity against the worm. H. B. Penfold, M. Phillips and I have found that cattle that have been artificially infected by drenching with *Tænia saginata* eggs go through a cycle of infestation with beef measles from which they recover when further drenching with the eggs will not give rise to another attack of the disease. They have, in fact, acquired a solid immunity against it. This may offer a basis for the control of the disease by vaccination.

Diphtheria Prophylaxis.

An effort has been made in Australia during many years to get rid of diphtheria. The remarkable success of W. H. Park in America and of many other workers in cutting down the incidence of and death rate from diphtheria stimulated us at the Commonwealth Serum Laboratories to prepare a toxin-antitoxin mixture suitable for the prevention of the disease.

This immunization against diphtheria is an extremely important part of our public health work. Before the Parliamentary Standing Committee of Public Works, when it was dealing with the School of Public Health, Sydney, I stated that diphtheria prophylaxis is carried out in practically all civilized countries and that inoculation is an effective preventive. This work should be without danger.

In Table III are set out diphtheria statistics for Australia and overseas countries. (Figures from the Report of the Parliamentary Standing Committee of Public Works, dealing with the School of Public Health, Sydney.)

TABLE III.

Diphtheria Statistics—Australia and Overseas Countries.

Notification Rate per 100,000 Population.		Death Rate per 100,000 Population.		Notification-Mortality Ratio (Deaths per 100 Notifications).	
Australia ..	152	North Ireland ..	10·75	Japan	24·5
New Zealand	149	Scotland ..	10·02	Bulgaria ..	15·0
Scotland ..	149	Canada	9·90	Canada ..	12·7
England and Wales ..	131	England and Wales ..	7·66	Irish Free State	12·2
North Ireland	101	U.S.A.	7·50	North Ireland	10·7
U.S.A. ..	79	Spain	7·10	U.S.A. ..	9·5
Canada ..	77	Irish Free State	6·33	Czechoslovakia	7·4
U.R.S.S. ..	65	Bulgaria ..	5·84	Scotland ..	6·7
Sweden ..	58	Japan	5·60	England and Wales ..	5·8
Austria ..	56	Australia ..	5·32	Germany ..	4·4
Irish Free State	52	New Zealand ..	4·05	Australia ..	3·5
Germany ..	48	Czechoslovakia	2·79	New Zealand ..	2·7

From this table we see that of all the countries dealt with the incidence of diphtheria is greatest in Australia—152 infections per 100,000 people. A convincing proof of poor prophylaxis! It is quite true that the notification mortality ratio is low and the deaths per 100,000 of population are low, but the high notification figures show that these results are not due to good prophylaxis. I would suggest rather that they are probably due to the use at an early stage of the disease of large doses of very potent serum (according to Scholes's scheme about 14,000 units per patient).

Conclusion.

I hold that we in Australia have splendid material out of which to develop research workers. We have an extremely sympathetic public who will supply us reasonably with money for research. Our weakness is that the majority of the curators of patronage have not been selected on one criterion only, namely, a long record of unselfish fruitful medical research.

Before closing, let me pay a tribute to Dr. J. V. Duhig. In 1920, when I was President of the Pathological Section of the Medical Congress at Brisbane, Dr. Duhig acted as Secretary. Since then we have been firmly associated in our desires for the promotion of medical research in this country. Dr. Duhig's own record of research as a voluntary worker, his sound ideas on the administration of national money for research, and his great interest in new scientific results fit him to advise this Branch on research matters. May I further express my indebtedness to another of your members, Dr. A. C. F. Halford, the author of "Lister Redivivus". There is a great deal to be said for his thesis in favour of antisepsis rather than asepsis; and if I might be allowed to interfere in a local matter, I should like to make the suggestion that the Queensland effort to reduce maternal mortality should be directed along Dr. Halford's lines. His book is simply a research into the literature. We want more than that, and I should like this Branch to give him the chance to reduce the local maternal mortality according to strict Listerian principles. I should not be surprised if he did it.

In concluding I would remind you that our motto for the Commonwealth Serum Laboratories was "May we so love nature that we may be worthy of her confidences". I believe that confidences should go to the lover. The need for research is pressing. "The harvest truly is plenteous, but the labourers are few; pray ye therefore the Lord of the harvest, that he will send forth labourers into His harvest."

INTRABILIARY RUPTURE OF HYDATID CYSTS OF THE LIVER: REPORT OF FIVE CASES.

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THE finding of bile-stained, intact or collapsed, hydatid daughter cysts in the stools was regarded for long as the result of rupture of a hydatid cyst into the alimentary tract. As abdominal surgery progressed, however, it was realized that the presence of cysts in this situation was frequently associated with obstruction of the common bile duct, and the obvious deduction was made that an hepatic cyst had ruptured into the gall-bladder or extrahepatic ducts. It remained for the South American and French surgeons to emphasize the importance of intrahepatic and the comparative rarity of extrahepatic intrabiliary rupture of hepatic cysts. The frequency of this complication has been generally recognized during the past two decades. Dew[1] states that "some degree of intrabiliary rupture is the commonest complication of hydatid disease of the liver". Dévé,[2] on the other hand, regards intraperitoneal rupture as the most frequent complication. He gives the figure for intrabiliary rupture as 5% to 10% of all hepatic cysts. The surgical importance of intrabiliary rupture lies in its frequency, in the fact that it is often the first manifestation of hydatid disease of the liver, in its close simulation of other lesions of the biliary tract, with consequent diagnostic mistakes, and in its ofttimes serious consequences.

CASE HISTORIES.

Reports of cases are of value in providing statistics, and also in illustrating problems in diagnosis and management. As more cases are reported, we obtain an idea of the true incidence of the various complications and the results thereof, and this gives a better understanding of hydatid disease generally. The following cases are reported because they illustrate most of the problems associated with the diagnosis and management of this particular complication.

CASE I was one of multiple hydatid cysts of the liver, one of which, a huge subphrenic cyst, ruptured into the biliary passage.

C.N., a male, aged fifty-six years, a labourer in the sheep districts around Junee, New South Wales, was admitted to hospital on February 4, 1935, with a history of recurring attacks of biliary colic with vomiting and jaundice during the previous thirty-seven years. The present attack had lasted six days and was longer in duration and more severe than any previous attack. The pain was severe, paroxysmal in nature, lasted for five to six hours, and was accompanied by much vomiting. An urticarial eruption usually, but not invariably, accompanied the attacks, preceding the pain by three to four hours. On two or three occasions he had vomited up small white bladder-like bodies. Jaundice invariably accompanied the attacks. The intervals between the severe attacks lasted two to four years, during which time the patient felt quite well. There was no history of flatulent dyspepsia or persistent gastric symptoms. Sometimes following these attacks the patient observed "white dots" in his fæces. He had noticed bulging of the right costal margin for the past twelve months. He had been admitted to the Royal Prince Alfred Hospital, Sydney, in 1913, with an abdominal swelling in the left flank, diagnosed as hydatid. He refused operation and since then the swelling had diminished in size.

On examination on February 4, 1935, his temperature was 36·9° C. (98·4° F.), his pulse rate 110 per minute, and his respiratory rate 25 per minute. He exhibited a slight icteric tinge, and there was an obvious bulging of the right costal margin. There was a swelling in the right half of the abdomen, which was firm and tender, and which moved on respiration. Dulness over the swelling was continuous with hepatic dulness. Upwards and to the left the area of dulness became continuous with the splenic dulness and with a dull area in the left hypochondrium. The upper border of the liver was elevated to the fourth intercostal space in the right mid-clavicular line. There was no evidence of pleural effusion. The fæces were clay coloured and pieces of laminated membrane and large collapsed daughter cysts—some over 2·5 centimetres (one inch) in diameter—and many small intact cysts were found. The urine contained bile pigments.

The Casoni test gave a positive reaction. A plain X ray examination revealed elevation of both cupolæ of the diaphragm, more so on the right side. Two calcified hydatid cysts, each apparently about 5·0 centimetres (two inches) in diameter, were seen, one in each lobe of the liver.

Operation was undertaken and the findings may be summarized as follows:

1. The anatomical relations in the right upper quadrant of the abdomen were grossly upset.
2. A calcified hydatid cyst was found on the anterior surface of the left lobe of the liver.
3. A huge subphrenic hydatid of the right lobe of the liver had ruptured into the right intrahepatic ducts.
4. A hydatid cyst was present in the left hypochondrium (? splenic in origin).
5. Cirrhosis of the liver was present.

A right paramedian incision was made. There was an obviously degenerated and partly calcareous thick-walled

cyst at the junction of the right and left lobes, and there was gross hepatomegaly with downward displacement of the liver—obviously produced by a second cyst. It was difficult at first to decide which cyst was at fault. A lax pouching of the wall of a cyst was felt where the lateral sector of the diaphragm met the supero-lateral surface of the liver (see Figure I). This at once gave the clue to which cyst had ruptured. It was a subphrenic hydatid of the right lobe with partly calcareous adventitia and which had already evacuated a quarter to a third of its contents, producing a lax pouch at the point marked "X" in Figure I.

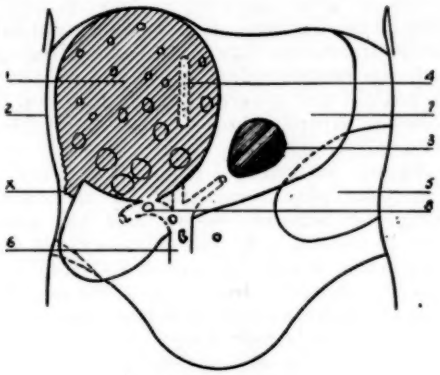

FIGURE I.

Schematic representation of operation findings in Case I. 1: Large subphrenic cyst of right lobe of liver. 2: Bulge of ribs. 3: Calcified cyst in left lobe. 4: Incision in upper part of cyst. 5: Cyst in left hypochondrium. 6: Duct into which cyst ruptured. 7: Left lobe, showing compensatory hypertrophy. 8: Common bile duct. X: Lax pouching of cyst wall.

The abdominal incision was prolonged up to the costal margin. The falciform ligament was cut. The hand was inserted between the liver and the diaphragm, and downward traction was made on the liver. A tumour, semi-fluid in consistency, was felt to be displacing the liver downwards and to the right. The abdominal cavity was packed off. An opening was made in the lower portion of the cyst with Kocher's forceps, and the cyst was evacuated by means of sucker, spoon and fingers. Four to five pints of bile-stained fluid, laminated membrane and several hundred daughter cysts, varying in size from a few millimetres to several inches in diameter, were evacuated. It was obvious, from the amount of bile found, that this cyst had undergone partial evacuation through the right hepatic ducts. The cyst was then washed out with saline solution and cleaned out as completely as possible with a sucker. A wide rubber drainage tube (2·0 centimetres in diameter and 15·0 centimetres in length) was inserted into the cyst and the wound was closed. In view of the poor condition of the patient and the grossly disturbed anatomy of the *porta hepatis*, no attempt was made to explore the common bile duct, as it was considered that, as the patient was not at the time suffering from common duct obstruction (normal stools and no jaundice), the duct would be kept clear if the cyst was evacuated. That this was justified was shown by the subsequent course of the case.

After the operation, collapsed and intact cysts (from a few millimetres to 2·5 centimetres in diameter), hydatid membrane, and foul-smelling yellowish-green fluid came through the drainage tube. Culture revealed *Staphylococcus aureus* and non-hæmolytic streptococci as the infective organisms. Some collapsed cysts were passed *per rectum;* but as there was no recurrence of biliary colic it is probable that they had entered the bowel before operation. The patient's stools regained their normal colour a few days after operation and remained so until his discharge from hospital. The size of the cavity gradually diminished, as indicated by X rays following the injection of 12·5% sodium iodide solution. The cavity was washed out with saline solution and then aspirated twice a day, and the sinus closed three months after the operation. The patient was troubled by persistent hiccup during the first week. Towards the end of his convalescence (eighth week) the patient's ankles began to swell; but this soon subsided. He was advised to return and have the cyst on the left side operated upon at a later date.

This patient had multiple hydatid cysts of the liver, including a huge subphrenic cyst, as well as a cyst arising from the region of the spleen. He gave a history of prolonged contact with sheep and dogs, having worked all his life in the vicinity of Junee. Considering the size of the subphrenic cyst, he must have been infested early in life, because hydatid cysts grow very slowly, especially in the liver, and are almost always approximately as old as the patient himself. The pain was typical of biliary colic and was due to the passage of daughter cysts, laminated membrane and *débris* down the intrahepatic and extrahepatic ducts. Vomiting is usual in these cases; but in this case there were sometimes small bladder-like bodies in the vomitus, these being due to regurgitation of duodenal contents, including small daughter cysts, through the pylorus. The jaundice was transient and not very deep, as the obstruction was intermittent. The attacks were accompanied by anaphylactic urticaria, described by the patient as "hives". This occurred very soon after the absorption of fluid from broken daughter cysts, and preceded by three or four hours the pain occurring when the collapsed cysts entered the ducts.

The combination of hepatomegaly, jaundice and biliary colic in this case was strongly suggestive of hydatid disease, and so the stools were successfully examined for daughter cysts and laminated mebrane, and a Casoni intradermal test was performed with positive results. The bulging of the ribs is more a feature of pulmonary hydatid than hepatic hydatid, and, in this case, was probably due to direct pressure of the very old-standing subphrenic cyst, which had displaced the liver downwards and rotated it to the left. The patient was very thin and slightly cachectic. This was probably due in part to failure of liver function—the bulk of hepatic functioning tissue had been destroyed—and possibly, in part, to hydatid cachexia, as described by Duval, McLaurin and others, following long-standing absorption of hydatid products.

Radiographical examination revealed elevation of both cupolæ of the diaphragm, the right more than the left. At operation, a subphrenic cyst was found, accounting for the displacement of the right cupola of the diaphragm upwards and the liver downwards. The cyst in the right lobe of the liver was huge and had destroyed much of the tissue of the right lobe; but the left lobe was found to be much increased in size. Hypertrophy of the left lobe is frequently found in extensive hydatid disease of the right lobe, being a compensatory change—a successful effort at maintaining hepatic function, and evidence itself of long-standing disease. Sometimes in such cases the left lobe is almost as large as a normal right lobe. This increase in size of the left lobe will explain the elevation of the left cupola of the

diaphragm. Against the downwards pressure by the liver counter-pressure was exerted upwards by the cyst attached to the spleen. The rotation of the liver downwards and to the left by the right subphrenic cyst, bringing the left lobe of the liver up against the left cupola of the diaphragm, is another factor in elevating this cupola.

The anatomy is often grossly distorted in these cases. The falciform ligament went to the left of the mid-line, while the liver overhung the region so much that the gall-bladder and common bile duct were not visualized. In addition, there were a great number of adhesions between the under-surface of the liver and the colon and duodenum, which were not exposed. The calcified cyst illustrates one end result of these cysts and goes to show what a variety of hydatid pathology can be found in the one patient. It is possible that this cyst, which must at one time have been very large, was the cause of the swelling in the left side recorded when the patient was in the Royal Prince Alfred Hospital in 1913. The old history papers indicate that it was much larger than at the present time. It is probable that this cyst too had leaked into the ducts with partial evacuation and natural cure, and it may well be that this was the cause of the earlier attacks of colic and the long periods of freedom. The outstanding feature of the case, then, is the long history during which fractional evacuation of one or possibly two hydatid cysts took place, with attacks of biliary colic. The cyst in the left flank was not defined or opened at operation, so that the pathological changes which it had undergone are unknown.

A very useful method of observing the rate of diminution in size of the cysts after operation, and also of determining the patency of the biliary ducts, including the common bile duct, namely, the introduction of a 12·5% solution of sodium iodide into the cyst, plugging temporarily, and then making a series of X ray examinations, was used to follow up the progress of this case. It is advisable to wash the cavity out afterwards, as the patient may, as in this case, develop iodism. The iodide probably had some beneficial effect, as the discharge was cleaner and the patient felt much better for it. Zwar[3] reported similar improvement in one of his cases. It is possible also that the beneficial effect follows the flushing out of infective hydatid *débris* which has accumulated in the biliary passages.

The delay in the obliteration of the cyst was due to the thick adherent adventitia, a direct consequence of the age of the cyst. Such cysts are often unable to collapse readily. They are frequently loculated; pouching, owing to irregular contraction after evacuation, is common, so that drainage is often inadequate and the sinus may persist for a long time, although drainage can be helped greatly by irrigation and by not shortening the tube too quickly. The swelling of the ankles was thought to be due to pressure on the inferior *vena cava*, as it appeared in a patient confined to bed who had no renal or cardiac disease, and in whom a large cyst existed in close proximity to the *vena cava*. The cyst in this case had undergone a further complication, namely, suppuration. An interesting feature of the suppuration in these cases seems to be that after open drainage the conditions within the cavity become aerobic with a change in the bacterial flora. At times this enables streptococci, which have been latent in the cyst, to multiply actively and so induce grave toxæmia, sometimes with death after a few days. As there was drainage at first through the ducts, and later through the tube, there was no great increase in tension, and there was very little toxæmia. It is also of great interest in that it illustrates how, if the offending cyst is drained, the common duct often looks after itself.

This patient has lowered hepatic function due to compression atrophy and cirrhosis, and his slow recovery after operation may be due to this. Regeneration, an inherent property of liver tissue, may in time play some part in the future in restoring the hepatic reserve and his metabolic function to normal. The abdominal cyst is unlikely to cause much trouble because of its situation; but he has been advised to have it operated on. Reports received six months after operation show that he is much better and is regaining his health so that he can undertake some of his former activities.

CASE II.—The second case was one of hydatid cyst of the liver, in which a mistaken diagnosis of gall-stones had been made and unnecessary cholecystectomy had been performed, followed by rupture of the common bile duct and formation of an extrahepatic collection of hydatid material and bile.

H.S., a male, aged fifty years, was admitted to hospital with biliary colic, vomiting and deep jaundice. He gave a history of twelve years' duration, during which time he had had recurring attacks of pain in the right hypochondrium, often lasting forty-eight hours, and recurring after intervals of three to six months. The attacks had increased in severity and frequency during the previous three months. The pain was very severe and would double him up. He would vomit three or four times during each attack. There was no history of urticaria or flatulent dyspepsia. Cholecystectomy had been carried out two years previously for suspected gall-stones without relief, while choledochotomy had been fruitlessly performed one month before admission, by the same surgeon at another hospital, for suspected stone in the common bile duct. He had been very ill since the last operation.

On examination the temperature was found to be 38·3° C. (101° F.), the pulse rate 110, and the respiration rate 25 per minute. The patient was deeply jaundiced and obviously very ill. Examination of the abdomen revealed the scar of a right paramedian incision. Deep to this scar was a large tender mass of a doughy consistency. Murphy's sign was present. No enlargement of the liver or spleen was detected. The urine contained bile and the stools were clay-coloured. A plain radiograph did not show any abnormality in the thorax or the hepatic region. The pre-operative diagnosis was suppurative cholangitis, with the rider that it might be suppurating hydatid cyst. The case was treated as urgent and no immunological tests were carried out.

On June 14, 1934, an incision was made in the previous operation scar and a dense mass of adhesions was at once encountered. The anatomical relations were grossly upset. The transverse colon was adherent to the anterior abdominal wall, and the stomach and duodenum to the liver. As the gall-bladder had been removed, an important landmark had been lost. The adhesions were separated and a collection of bile and hydatid daughter cysts was found, walled off from the general peritoneal cavity by omentum. The foramen of Winslow was obliterated by adhesions, and so the lesser sac was unaffected. The bile

and daughter cysts were removed by suction and the extrahepatic situation proved. The ruptured common bile duct was made out in the posterior wall of the collection, where it appeared like a white ribbon. By means of a sucker and a rubber catheter, the larger cavity and the common bile duct were emptied of daughter cysts and membrane. The catheter was passed up and down the ducts, and they were completely cleared of *débris*. The liver was then explored with needles to determine the site of the cyst. This was found to be deeply situated in the right lobe, thick-walled, and apparently empty and collapsed, and so it was left undrained. A large drainage tube was inserted down to the extrahepatic cavity and the abdomen closed. (See Figure II.) The tube drained bile and daughter cysts for one month, the stools remaining clay-coloured. Gradually the stools regained their normal colour and the biliary fistula closed. It is interesting to note that the student dresser discovered collapsed bile-stained cysts in the stools in the first motion after the operation. The Casoni intradermal test was also found to be positive at this time.

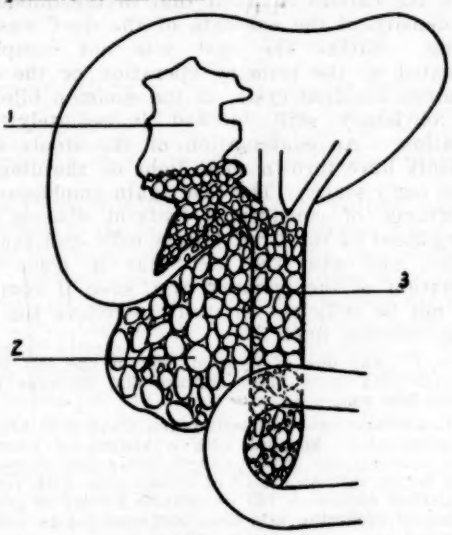

FIGURE II.

Diagram showing conditions presented by Case II. 1: Incompletely collapsed cyst containing some daughter cysts. 2: Markedly dilated and ruptured common bile duct. 3: The extrahepatic collection of cysts, bile and *débris*.

As the patient showed some signs of persistent toxæmia, a transpleural approach to the cyst was attempted on July 20, 1934. A rib was resected. There were no pleural adhesions. The diaphragm was covered with dilated veins, suggesting an underlying hydatid. The diaphragmatic surface of the liver was explored with needles. A thick-walled cavity containing some bile and some inspissated *débris* was found with a needle. Owing to the absence of pleural adhesions to the depth of the cyst, and to the fact that it was considered to be empty, the cyst was not drained by the transpleural route. Lipiodol was injected into the cyst cavity and X ray examination later showed that this was scattered through the bowel, proving communication with the biliary tree and bowel.

This patient had a hydatid cyst of the right lobe of the liver, which had ruptured into the biliary tree. The long history of recurring attacks of colic indicates that the cyst was undergoing intermittent evacuation, the communication with the biliary tree becoming alternately blocked with *débris* and unblocked. The sequence of events was as follows:

1. Intrabiliary rupture of a cyst leading to the mistaken diagnosis of cholelithiasis, though there was no history of flatulent dyspepsia, and hydatid disease was not excluded nor apparently even considered. Not only was the cholecystectomy which was performed without avail as regards the symptoms, but an important landmark was removed, rendering the later operative treatment difficult.

2. Intermittent drainage of the cyst led to further attacks of colic and jaundice, so that a residual stone in the common duct was suspected. Choledochotomy was performed without revealing anything abnormal (probably because it was carried out during a non-icteric interval) and without any beneficial result. This temporarily relieved the tension in the biliary tree; but when the communication between the cyst and duct became patent once again, *débris* and cysts passed down the common bile duct, which again became blocked and dilated. The tension caused a large rupture at the site of the previous choledochotomy, and resulted in a localized choleperitoneum with a collection of cysts and *débris* in the region of the *porta hepatis*. The relief of the tension in the common bile duct and reestablishment of the flow of bile resulted in amelioration of symptoms; but the introduction of infection rapidly supervened and the patient on admission was extremely ill, presenting the signs and symptoms of suppurative cholangitis. Two mistakes in this case were: (*a*) failure to examine the stools for daughter cysts and hydatid membrane; (*b*) failure to carry out a Casoni test, either of which would have established the diagnosis.

At operation the localized choleperitoneum was drained, the common bile duct being located with difficulty, as the gall-bladder—the most important landmark in this region—had been removed. This illustrates how an incorrect diagnosis may lead to incorrect surgery, which may in turn increase the difficulties encountered at subsequent operation. The decision to leave the offending cyst undrained, because it was believed that it was completely evacuated as the result of the findings of exploratory puncture, seems to have been justified, as the patient, when seen fifteen months later, was in perfect health, carrying on his full work as a boat builder. This experience also illustrates that if a tubular structure is left widely open in a ribbon form, it will in time be reconverted by the influence of the surrounding tissue reaction into a tubular form again.

CASE III.—The third case was one of hydatid cyst of the liver, which had ruptured into the biliary passages, with effectual evacuation of the cyst at operation, but persistence of common bile duct block.

J.L., a male, aged forty-one years, who had been associated with sheep all his life, was admitted to hospital complaining of biliary fistula, jaundice and clay-coloured stools for the previous ten months. About fifteen years previously the patient had had an attack of pain with vomiting and jaundice lasting two days. Then, four years later, he had a similar attack and was seen by a doctor who told him he had an enlarged liver. About three years before admission to hospital the patient fell on his back and developed acute epigastric pain with vomiting and jaundice, lasting five days. Two years later he had a similar attack and was operated on in a country hospital for a large hydatid of the liver. This was drained and, after being in bed

for seven weeks, he got up, but had a recurrence of pain and vomiting. During the ten months prior to admission bile had been draining intermittently from the operation wound. During this time he had a definite icteric tinge and lost 31·5 kilograms (five stone) in weight.

Examination revealed the patient to be very jaundiced and emaciated. His temperature was 36·9° C. (98·4° F.); his pulse rate was 100 and his respiratory rate 25 per minute. Examination of the abdomen showed a biliary fistula at the site of an old wound in the right hypochondrium. The right lobe of the liver was enlarged and firm, but not tender. No other systems were affected. The stools were clay-coloured, but no daughter cysts were found therein.

Radiographical examination, including screening of the diaphragm, revealed nothing abnormal.

The pre-operative diagnosis was persistent obstruction of the common bile duct due to intrabiliary rupture of a hydatid cyst of the liver.

Operation was performed on July 23, 1931. As the gall-bladder had not been removed, the common bile duct was readily found. It was greatly distended, its diameter being almost equal to that of the duodenum. The contents of the duct seemed to be very hard. Choledochotomy was performed and the dark bile-stained *débris* removed. This was found to contain much hydatid membrane, set in a matrix of dark inspissated bile. By means of a scoop, followed by the use of a catheter and suction, the ducts were completely cleared to the satisfaction of the operator. A drainage tube was put into the common duct.

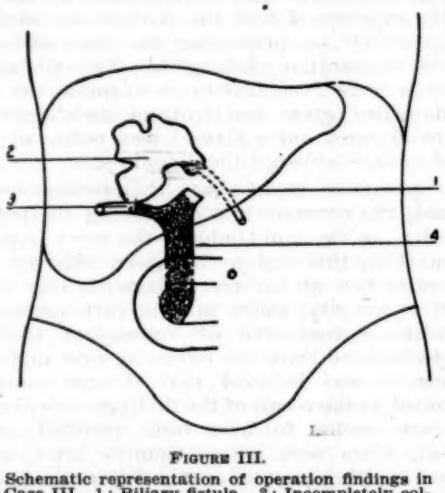

FIGURE III.

Schematic representation of operation findings in Case III. 1: Biliary fistula. 2: Incompletely collapsed, loculated, thick-walled cyst. 3: Opening of cyst into right duct. 4: Distended common bile duct full of cement-like *débris*.

The biliary fistula closed within two weeks, during which time the jaundice disappeared, the stools regained their normal colour, and the patient put on weight. He was discharged, but readmitted five months later with another attack of biliary colic, with vomiting, clay-coloured stools, and jaundice. This must have been due to some residual *débris* in the intrahepatic ducts passing down; but it soon settled down and the patient was discharged six days later, the jaundice having rapidly disappeared. Since that time (four years ago) the patient has not had any further trouble as far as can be ascertained, and he reports himself as normal in health and activity.

A feature of this case is again the length of the history of intermittent evacuation of cysts through the biliary passages. The occurrence of hepatomegaly, associated with attacks of jaundice and biliary colic, should have suggested the diagnosis of hydatid of the liver eleven years ago, the more so because the patient was a male and had spent most of his life in contact with sheep. The history of trauma is a feature which was absent in the other cases. The trauma was indirect, the patient having fallen on his back, and though it may have precipitated a further passage of hydatid material, it may have been only a coincidence. The persistent biliary fistula, with intermittent drainage, resulted from the same causes as in Case II, the distal end of the common bile duct being blocked with hydatid *débris*, drainage taking place through the cavity of the now empty original cyst. At operation choledochotomy was facilitated by the presence of the gall-bladder.

The degree of distension to which the common bile duct may reach is well illustrated in this case, in which the calibre equalled that of the duodenum. The density of the contents of the duct was surprising. Either the cyst was not completely evacuated at the primary operation or the ducts contained residual cysts, as the common bile duct was obviously still blocked immediately after operation. An examination of the stools would certainly have thrown some light on the diagnosis in the early stages. The case again emphasizes the importance of considering hydatid disease when enlargement of the liver, biliary colic and jaundice coexist, and also the fact that in some cases evacuation of the primary cyst, even if complete, may not be sufficient in itself to relieve the block of the common duct.

CASE IV was one of hydatid cyst of the left lobe of the liver with intrabiliary rupture and blockage of the common bile duct.

F.H., a female, aged forty-one years, a native of Armidale, was admitted to hospital with a history of attacks of biliary colic for the previous three and a half years. The illness began with an attack of biliary colic with vomiting and marked flatulence, but no definite history of jaundice. Because of recurring attacks a cholecystectomy had been performed six months later. The patient was quite well for three months, then again began to suffer from attacks of biliary colic similar to the initial attack, but increasing in severity and duration, with varying degrees of jaundice. There was no mention of urticaria in the history.

Physical examination revealed that the temperature, pulse and respiration were normal. The patient was jaundiced and had obviously lost weight. The abdomen revealed an old scar of a right upper paramedian incision. There were tenderness and rigidity in the right hypochondrium. The urine contained bile. There was no report of an examination of the stools. Radiographical examination failed to reveal any abnormality in the thorax or abdomen.

At operation the findings were as follows: (i) adhesions between the duodenum and fossa for the gall-bladder; (ii) the common bile duct dilated and filled with daughter cysts; (iii) a multivesicular cyst of the left lobe of the liver abutting on its inferior surface.

A mid-line incision was made, the adhesions between the duodenum and liver were divided, and the distended common bile duct was exposed. The common bile duct was needled and yellowish fluid and hydatid *débris* were aspirated. Choledochotomy was performed and daughter cysts of varying size, hydatid membrane and *débris* were removed. The liver was then examined and a cyst found subperitoneally, situated in the left lobe of the liver. This was incised and evacuated; its wall was white and thick, and not very tense, and it was regarded as the offending cyst. The common bile duct and hepatic cyst were then drained with rubber tubes and the wound was closed.

Whilst recovering from the influence of the anæsthetic the patient vomited fluid containing several daughter cysts. The convalescence was uneventful. After draining bile for a fortnight the fistulæ closed. Since discharge the patient has been quite well.

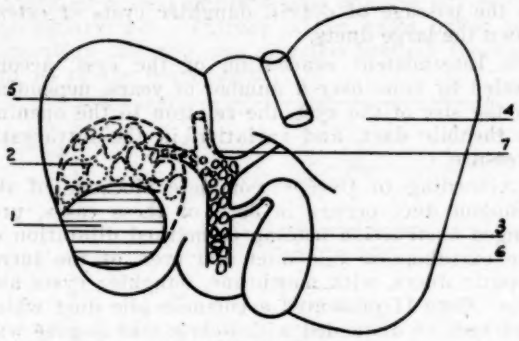

FIGURE IV.

Operation findings in Case IV. 1: Cyst abutting on inferior surface of the liver. 2: Portion of cyst deep in the left lobe. 3: Common bile duct filled with daughter cysts *et cetera*. 4: Right lobe of the liver. 5: Left lobe of the liver. 6: Gall-bladder. 7: The left hepatic duct, into one branch of which the cyst ruptured.

Case IV is of interest in that the patient was the only female in the series. Her age corresponded with the age incidence of the disease and this complication. Contact with sheep and dogs was to be expected, since she had lived at Armidale for most of her life. The history of flatulent dyspepsia with attacks of biliary colic led to a diagnosis of cholecystitis, and so cholecystectomy was performed. An examination of the stools or a Casoni test would probably have revealed the real condition. The quiescent period of three months following the operation led both the patient and surgeon to believe that a cure had been achieved, only to be disillusioned by a recurrence of the colic with the same degree of pain as before operation.

CASE V.—L.A., a male, aged thirty-eight years, of an outlying district of Sydney, was admitted to hospital with a history of recurring attacks of biliary colic, with vomiting and jaundice, over a period of five years, culminating in a severe attack of one week in duration, and characterized by attacks of colic during the intervals, between which there was more or less constant pain, with very deep jaundice. There was no history of flatulent dyspepsia, urticaria or close contact with dogs.

The patient's temperature was 37·8° C. (100° F.), the pulse rate 90 and the respirations 25 per minute. He was mildly jaundiced. There were tenderness and rigidity in the right hypochondrium; but no hepatomegaly or signs in the thorax were detected. The urine contained bile; but there was no report of any examination of the stools.

A blood count revealed a leucocytosis of 14,400 and an eosinophilia of 29%. The Casoni reaction was positive. Radiographic examination revealed nothing abnormal in the chest or the abdomen.

The case was regarded as a hydatid cyst of the liver, almost certainly in the classical site beneath the right cupola of the diaphragm, which had burst into the biliary passages, with obstruction of the common bile duct and mild cholangitis.

A portion of the eleventh rib was resected in the mid-axillary line. The pleura was retracted upwards, the diaphragm was incised and retracted, and a large subphrenic cyst was found. This was incised and proved to be an old cyst, as it was multivesicular and had a very thick wall. The contents were evacuated and the cyst drained.

The common bile duct unblocked itself, the stools regained their normal colour, and the jaundice gradually disappeared. The convalescence was interrupted by lobar pneumonia, from which the patient recovered. The biliary fistula closed within three weeks and the patient was discharged. As far as can be ascertained, there has been no recurrence of symptoms.

This case resembled the others in that the patient was a male, and his age corresponded to the age incidence of this complication. The jaundice in this patient was probably due to a combination of obstructive and infective causes. He had cholangitis, as indicated by the pyrexia, leucocytosis and common bile duct obstruction. In this respect he resembled the patient in Case II. The pain in this case was of two types: the intermittent colicky type and a constant aching pain present between the attacks, due to the cholangitis. The importance of a blood count is shown in this case. The eosinophilia, associated with colic and jaundice, suggested a Casoni test, the result of which proved to be positive.

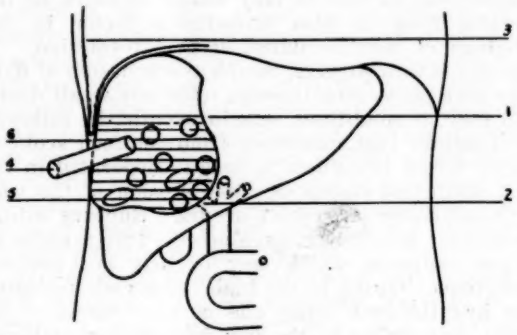

FIGURE V.

Diagram showing operation findings in Case V. 1: Partially evacuated subphrenic cyst. 2: Communication between cyst and duct blocked by a daughter cyst. 3: Parietal pleura retracted in costo-phrenic angle. 4: Drainage tube. 5: Diaphragm. 6: Costo-phrenic space, normally lined by parietal pleura and across which the cyst was drained.

Cases IV and V are chiefly of interest from the point of view of treatment. In Case IV the diagnosis was uncertain, whereas in Case V it was practically certain, and a different method of treatment was carried out in each case. In the former a laparotomy was done and a choledochotomy performed. When it was obvious from the contents of the common bile duct that hydatid disease was present, the liver was explored. The cyst was located in the left lobe of the liver, abutting on the peritoneal covering of the inferior surface, and so readily accessible by the abdominal route. It was evacuated and drained. In Case V, as the cholangitis was only mild, the jaundice was subsiding, and the general condition of the patient fairly good, the operation of election was carried out. A transcostal approach was made and the cyst itself attacked, leaving the common bile duct to unblock itself. The result justified the method.

Again in Case IV the difficulties arising from a previous cholecystectomy are illustrated; not only is an important landmark lost, but adhesions increase

the duration of the operation. The unusual site of the cyst in this case is a very interesting feature. Only a very low percentage of cysts of the left lobe rupture into the biliary passages, as will be shown later. Case V, too, illustrates a not infrequent complication of operations in the subphrenic region, namely, pneumonia.

DISCUSSION.

Pathology.

Biliary Contamination.—As the hepatic cyst grows, it increases in size at the expense of the hepatic tissue, which undergoes quiet pressure necrosis. The bile duct epithelium is more resistant than the cells of the hepatic lobules and the ducts may become incorporated in the adventitia. Eventually the smaller ducts are eroded or give way, so that the biliary leak may occur, and the cyst becomes contaminated with bile—a common finding in cysts of any appreciable size. This biliary contamination, in that it may induce changes in the hydatid fluid, is also probably a factor in the ætiology of reactive daughter cyst formation. If the cyst is univesicular, complete evacuation of fluid may take place, even through relatively small ducts, and may, if conditions remain aseptic, be followed by daughter cyst formation from retained scolices. Later, larger bile ducts become incorporated in the cyst wall, and sooner or later erosion of the wall of a relatively large duct occurs. Rupture into a large duct is nearly always preceded by a series of minor ruptures, which may or may not produce symptoms. Owing to the high intracystic pressure, the hydatid cyst being one of the tensest of all cysts, and owing to the low intrabiliary pressure, the cyst contents burst into the biliary passages. If the amount of material passing into the hepatic ducts is small, because of rapid occlusion of the opening with hydatid *débris*, varying anaphylactic symptoms followed by transient colic with jaundice will occur. The biliary tree will evacuate itself in a few days, the symptoms will disappear and the patient seem to be in perfect health once again, until contraction of the adventitia, the onset of suppuration or some other factor causes such increase in intracystic tension that another rupture takes place. If much material escapes, as is often the case with a large cyst, the intrahepatic and extrahepatic ducts may become full of daughter cysts, laminated membrane and *débris*, until the pressure behind the ampulla of Vater reaches a level sufficient to open it. It would seem that the sequence of these mechanical events may well be summarized thus:

1. A series of minor biliary leaks inside the adventitia, with staining of cyst contents and possibly alteration of nutritive conditions inside the cyst.

2. Intrabiliary rupture of univesicular cyst with anaphylaxis and mild biliary colic and temporary reduction in the size of the cyst, followed in some cases by death of the parasite, inspissation, and enclosure of fibrotic tissue.

3. Or daughter cyst formation, the cyst gradually increasing in size so that it reaches its original size again in ten to twenty years.

4. Secondary intrabiliary rupture with colic due to the passage of *débris*, daughter cysts *et cetera* down the large ducts.

5. Intermittent evacuation of the cyst, accompanied by colic over a number of years, depending on the size of the cyst, the relation to the opening in the bile duct, and variation in the intracystic pressure.

According to Dévé,[2] complete blockage of the common duct occurs in 30% of these cases, prolonged obstruction leading to marked dilatation of the extrahepatic bile duct and even of the intrahepatic ducts, with membrane, daughter cysts and bile. Case II presented a common bile duct which had been so distended with *débris* that it gave way at a weak spot, while in Case III the calibre of the common duct equalled that of the duodenum. In the majority of cases (60%) the gall-bladder also becomes distended with bile and hydatid material, owing to retrograde passage of daughter cysts and membrane up to the cystic duct. This led early observers to believe that rupture into the gall-bladder was a relatively common complication of hepatic hydatid disease. This also explains the origin of gall-stones in some cases of intrabiliary rupture, hydatid *débris* apparently acting as nuclei for the deposition of cholesterol and pigments. Owing to the thickness of the adventitia in old cysts, the collapse is usually incomplete, resulting in inefficient drainage, with a long history of intermittent evacuation; suppuration is an ever-present risk.

The Site of Rupture.—Rupture may occur into the intrahepatic or extrahepatic ducts or into the gall-bladder. Intrahepatic intrabiliary rupture is the more important type. Proof of communication between the cyst and the biliary tree has been established by: (i) injection of lipiodol into the subphrenic cyst and its detection radiographically in the bowel later, as in Case III; (ii) injection of sodium iodide solution (12·5%), which will outline the cavity and in some cases enables the common duct to be visualized, as in the case reported by Zwar[3] (an unsuccessful attempt was made to do this in Case I); (iii) the frequency with which bile runs into the cyst after evacuation at operation.

The most common site of the offending cyst is in the right lobe of the liver, towards the subphrenic surface. In some cases no cyst can be found on the external surface or in the liver by needling, and in such cases a cyst deeply situated in the centre of the liver is probably at fault. Before it was realized that a subphrenic cyst was the usual source of trouble, and as no cyst was visible at laparotomy, probably too many cases were regarded as arising from rupture of a central cyst.

The French and South American writers gave the relative frequency of rupture as 11% to 16% into the extrahepatic ducts and gall-bladder (5%), and 84% to 90% into the intrahepatic ducts. Since

the majority of hepatic cysts occur in the right lobe of the liver, it follows that intrabiliary rupture occurs most commonly into the right half of the biliary tree (55% to 60% of all cases). In 25% to 30% of cases rupture occurs into the left half of the biliary tree. In four of the cases under consideration the cyst was situated beneath the right cupola of the diaphragm.

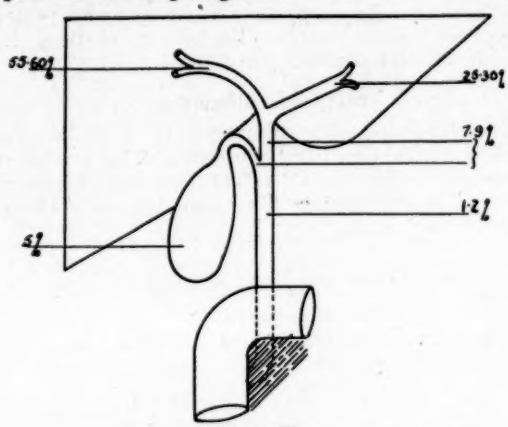

FIGURE VI.
Schema after Dew,[3] showing sites of rupture.

According to Finochietto,[4] 60% of offending cysts are situated postero-latero-superiorly, 20% in the antero-superior portion of the right lobe, and 10% in the inferior portion of the right lobe. The other 10% of cases are cysts situated in the left lobe; but these often burst into the right half of the biliary tree. Thus in the majority of cases the cysts are situated in the right lobe of the liver and are subphrenic in position. The course of intrabiliary rupture depends to some extent on the size of the opening into the duct and its relation to the cyst. If the opening into the duct is large and situated in the lower or dependent part of the cavity, it is obvious that drainage and evacuation will be much more efficient.

The Type of Cyst.—The cyst at fault may be univesicular or multivesicular. Usually it is multivesicular, showing daughter cyst formation following a series of minor leaks and culminating in frank intrabiliary rupture with severe colic. Dévé[5] states that in 90% of these cases the cyst is multivesicular.

With multivesicular cysts, owing to the nature of their contents, intermittent fractional evacuation occurs. The smaller daughter cysts may be passed down the common bile duct and into the duodenum intact; but the larger ones rupture and are passed in the collapsed state.

Following rupture of a univesicular cyst, if complete extrusion of contents and good collapse of the cyst has occurred, natural cure by inspissation and fibrosis may result. As alternatives, daughter cyst formation will occur if conditions remain aseptic, while occasionally suppuration will take place. The intracystic pressure and the pressure of the head of bile behind the obstruction are the main factors in forcing the *débris* down the ducts, the passage of which is facilitated by the slippery nature of the membrane and daughter cysts.

Pathological Sequelæ.—Intermittent and variable obstruction of the ducts, which occurs in most cases, leads to chronic fibrotic changes in the liver and depression of hepatic function, particularly in long-standing cases.

Suppuration is the most important complication. Some degree of infection occurs in at least 75% of the cases of intrabiliary rupture and in at least 10% of cases is caused by anaerobes (Dew). It is particularly apt to occur in those cysts with bad drainage, where the wall is loculated, or is thick and will not collapse, and when the opening in the duct is unfavourably situated. Bad drainage results in stasis of dead material and retrograde passage of infection from the duodenum or infection from the bile. Suppuration of the hydatid is often associated with cholangitis and cholecystitis, and sometimes with hepatitis. A subphrenic abscess is a possible but rare sequel, as subphrenic cysts usually have dependent drainage and firm adhesions are formed, obliterating the space between the liver and the diaphragm. When infection occurs, the toxæmia is in direct proportion to the tension within the cyst, and is greatest when gas-forming anaerobes are present. This is a very serious complication. It at once modifies the clinical picture, and such cases carry a comparatively high mortality; indeed they are responsible for most of the deaths in hepatic hydatid disease.

According to Dévé, spontaneous cure occurs in 24% of cases. This is obviously more likely to occur in a univesicular cyst with dependent drainage. The cyst collapses completely and the adventitia undergoes fibrosis and even calcification. It may even occur with incomplete evacuation of the cyst, the inspissated laminated membrane lying crumpled up within the contracted adventitia. Such cases of natural cure are not uncommonly found at autopsy, and specimens are featured in all museums.

As indicated above, gall-stone formation may occur around hydatid *débris* left in the gall-bladder—the *cholelithiase hydatique* of Dévé.

Explanation of Symptoms.

The effects of rupture may be summarized thus:

General effects:

1. Immediate—anaphylactic, usually manifested as urticaria.
2. Delayed—toxæmia due to infection, impairment of hepatic function.

Local effects:

1. Immediate—mechanical block associated with biliary colic and obstructive jaundice.
2. Delayed—suppuration.

Hydatid disease of the liver is practically symptomless until some complication occurs. Unless the cyst projects into the peritoneal cavity it often does not make itself manifest until relatively late in life, as complications usually occur only in cysts of moderately large size, as in these cases. The age incidence of intrabiliary rupture is 35 to 50 years.

Pain.—Pain is usually of dramatically sudden onset, though there is sometimes a history of trauma, as in Case III, either a fall or, in some cases, excessive muscular effort. There is often no history of premonitory symptoms, though some patients give a history of epigastric discomfort, hepatic pain or flatulence, probably the manifestation of minor biliary leaks, with low grade infection, and these may presage a frank rupture. Classically, the first symptom is pain of sudden onset, severe and colicky in nature, accompanied by sweating, vomiting and shock. It is not often as severe as gall-stone colic, because of the softness and slippery character of the hydatid material. In 60% of cases the pain, which is due to distension of the biliary passages, is repeated at varying intervals, due to intermittent evacuation, resulting in fresh masses of material passing down the ducts. Another factor in the causation of the pain is infection, as in Case V. This causes a constant aching pain as a rule, and is associated with hepatic tenderness and pyrexia. The patient often feels in perfect health between the attacks, as in Cases I and III. It is important to realize that this cessation of pain over long periods of years sometimes leads to the belief that the condition is cured and may also lead to mistakes with regard to treatment.

Urticaria.—Urticaria occurs in some cases, as in Case I. It is the result of an antigen-antibody reaction in a sensitized individual. Other manifestations of anaphylaxis may occur, such as cyanosis, dyspnœa, or cardio-vascular symptoms.

Jaundice.—Jaundice occurs in approximately 90% of cases. It depends on: (*a*) the degree of common duct block, (*b*) on the presence or absence of infection. When the block is of short duration, transient jaundice occurs; but when complete block occurs, intense jaundice follows. Infection, when present, is another factor in prolonging the jaundice. The jaundice is variable, but is rarely as intense as with stone in the common duct, and is often of days' or weeks' duration. It is seldom that jaundice is due to a cyst pressing on the common bile duct. As stated by Dew,[1] the association of hepatomegaly and persistent jaundice in this country is more likely to be due to hydatid disease than to gall-stones.

Vomiting.—Vomiting is a usual concomitant of colic. Sometimes, as in Cases I and IV, small white bladder-like bodies may occur in the vomitus, due to regurgitation of daughter cysts through the pylorus.

Fæces.—Examination of the fæces constitutes the most important source of information. Owing to the remarkably resistant nature of hydatid hyalin material, large collapsed daughter cysts or small intact cysts, bile-stained, with or without irregular pieces of hydatid membrane, are present in the fæces and can usually be found by simple washing, as in Cases I and II. If large intact daughter cysts be found, such as are too large for passage of the common duct, intra-alimentary rupture (into the duodenum or colon) must be suspected.

Hepatomegaly.—The increase in the size of the liver is due to downward displacement of the liver by a subphrenic cyst, compensatory hypertrophy, or both. If the upper border of liver dulness is also elevated, then there is almost certainly a subphrenic cyst, as in Case I. It must be borne in mind that if fractional evacuation has been taking place for a long time, the size of the cyst may be so reduced that hepatomegaly may be absent and the patient may even state that a preexisting swelling has become smaller or has disappeared.

Diagnostic Difficulties.

Cholelithiasis.—The commonest error in diagnosis is cholelithiasis with biliary colic. Most errors are made in males, as Cases II and III illustrate. Table I is helpful in differential diagnosis (Dew).

TABLE I.

Comparison of Main Features of Biliary Colic due to Hydatid Disease and to Gall-Stones.

	Hydatid Disease.	Gall-Stones.
1. Sex	M 1 / F 1	M 1 / F 4
2. Age	30 to 45	45 to 55
3. Pain	Moderate	Severe
4. Jaundice	Invariably present	Often absent
5. Liver	Frequently enlarged	Not enlarged
6. Stools	Daughter cysts and hydatid membrane often found	Gall-stones occasionally found
7. Casoni	90% positive. Complement fixation test usually positive in the other 10% of cases	Negative
8. X ray	Elevation of the diaphragm	No diaphragmatic distortion
9. Graham's test	Negative, except when there are associated gall-stones	May be positive
10. Previous history	Seldom any history of gastric disturbance	Frequently a long history of flatulent dyspepsia.

Occasionally gall-stones and hydatid disease coexist in the same patient, the latter in some cases possibly playing some part in the ætiology of the former. In all cases of apparent gall-stones in the male hydatid disease should be suspected. Whenever a case of biliary colic with jaundice, with or without hepatomegaly, presents itself, the stools deserve careful investigation and a Casoni test should be performed as a routine. Errors in diagnosis would be much less common if it were widely recognized that "hydatid cysts not infrequently and gall-stones comparatively rarely involve the large hepatic ducts" (Dew). The latency of the condition and the good general health associated with it are also important points to be considered in hydatid disease. When in doubt and laparotomy has been decided upon, it is advisable, too, to bear in mind that unless there is certainty as to the freedom of the common duct, cholecystectomy will be disadvantageous should the symptoms of colic recur and choledochotomy become necessary at a later date.

Other Causes of Hepatomegaly or Swellings in the Region.—Hepatomegaly in this country in an otherwise healthy individual should always arouse a suspicion of hydatid disease. Compensatory

hypertrophy of the left lobe of the liver has led to some erroneous diagnoses of *situs inversus* or cyst of the left lobe. Hepatomegaly has to be distinguished from an enlarged gall-bladder, from hydronephrosis, and, in some cases, from pancreatic cyst.

Dulness at the Base of the Right Side of the Thorax.—A subphrenic hydatid is sometimes difficult to distinguish from a pleural effusion. They coexist in 8% to 10% of cases of subphrenic hydatid cyst. Dulness situated low down over the right side of the chest, with biliary colic and jaundice, would almost certainly be hydatid. Examination of the patient in the knee-elbow position and comparison with the findings in the erect posture will sometimes, owing to the interposition and increase of pulmonary resonance, help greatly in the clinical detection of the subphrenic site of the cyst.

Suppuration.—When suppuration occurs, then cholecystitis, cholangitis and subphrenic abscess have to be excluded. Persistent jaundice with pyrexia should arouse a suspicion of a recent intrabiliary rupture of a hydatid cyst with infection. As the above conditions can at times accompany hydatid disease, differentiation becomes very difficult. It is important to realize that gas is sometimes found in these cases, owing to the activity of anaerobic organisms. In these cases the presence of a pneumocyst may be detected by the shifting resonance in the hepatic area and also at times by the detection of a fluid level, with gas above it, by radiography.

Management.

Investigation.

Stools.—Examination of the stools is the most important line of investigation. The presence of bile-stained membrane, which can be easily recognized, puts the diagnosis beyond doubt. It is so simple that it deserves a more important place. The bed-pan is put under a slowly running tap and the fæces are washed away gently, leaving the typical gelatinous membrane behind.

Immunity Tests.—The Casoni intradermal reaction is positive in 90% of cases of intrabiliary rupture. This reaction may occasionally be negative owing to desensitization following the sudden absorption of massive doses of antigen. In the remaining 10% of cases the response to the complement deviation test is almost invariably positive. Therefore in this condition one or other of the immunity reactions is positive.

Radiography.—X ray examination of the liver may show calcified cysts or liver cysts with some calcareous changes in their walls, as in Case I. Also in the case of many subphrenic cysts X rays may reveal elevation, distortion or diminished movement of the diaphragm, the presence of a gas bubble, or an effusion into the right pleural cavity.

Surgical Treatment.

Much controversy has been waged over the method of treatment in cases with biliary obstruction. The French mainly advocated clearing and draining of the biliary tree, thus abolishing the symptoms of obstruction and diminishing the risks of infection, leaving the cyst alone to evacuate itself over a number of years, or more often attacking it directly shortly after. South American and most Australian surgeons advocated a radical attack on the offending cyst, and drainage, leaving the biliary tree to look after itself, running the risks of infection. Geroulanos and Maccas[7] were of the opinion that a cure could be effected by either method, but that a combination of both procedures, either at the same time or as a two-stage procedure, was the ideal method of treatment. Dévé[2] in 1932 advocated drainage of the original cyst, unless the degree of biliary obstruction was marked, because he considered that the biliary passages usually became cleared of their obstruction spontaneously. This as a rule means a transcostal approach, because the cysts on the supero-lateral aspect of the liver are the cysts which most frequently evacuate themselves into the biliary passages. He considered that cysts on the inferior surface of the liver did not often undergo intrabiliary rupture, tending rather to rupture intraperitoneally. The reason why choledochotomy has been done primarily in so many cases is probably that either the condition was unrecognized before operation or the cyst at fault was not found at laparotomy, as it was subphrenic in position. Case I illustrates the difficulty in finding the cyst at fault if it is not realized that the offending cyst is probably subphrenic in position. Dew agrees with Dévé and the South American surgeons that the routine treatment should be to treat the cyst at fault, and further advises that the operation should be carried out when the jaundice has gone and the ducts are at least temporarily free. Choledochotomy may be a very difficult procedure where the gall-bladder has been removed, as it not uncommonly has been, owing to diagnostic error, some years earlier.

Although the five cases here recorded illustrate the various operative procedures which may be necessary, it is advisable to consider them in more detail.

Operative Procedures.—1. (*a*) If the diagnosis is uncertain or if, as often happens, the diagnosis of gall-stones is made, laparotomy may in the first place reveal an apparently normal biliary apparatus. This is likely to occur in those cases in which operation is performed during a free interval, when the ducts are clear of obstruction. In male patients the surgeon should suspect that there is a hidden hepatic hydatid and should look for hepatomegaly and search the right lobe carefully for a hydatid cyst, using a needle if necessary. It must be remembered that if the symptoms have been present for a long time the cyst may have lost its normally tense nature and be readily overlooked. If the cyst is readily accessible, it should be evacuated and drained. It is often high up in the right lobe and comparatively inaccessible by this route, but it may be exposed sometimes by extending the incision, dividing the falciform ligament and retracting the liver downward somewhat, as in Case I, or by

mobilizing the costal cartilages of the lower ribs and retracting the diaphragm and pleura after the method of Ivanissevitch.[6] If the patient's condition warrants it, a cyst inaccessible by this route should be drained transcostally in the usual way (Case V), care being taken to make the incision as far forward as possible in order to avoid pleural opening and possible contamination; for most, if not all, of these cysts are infected. The gall-bladder should not be removed, because it may be necessary at a later date to perform choledochotomy to relieve obstruction of the common duct, which has persisted in spite of drainage of the offending cyst, and the presence of the gall-bladder is a great help as a landmark. Although the diagnosis was certain, the above operative procedure was carried out in Cases I and V, and the decision not to open the common duct was justified by the fact that it unblocked naturally.

(*b*) The common duct and, indeed, the gall-bladder may contain hydatid membrane or cysts when operation is performed during the obstructive stage. This material is soft and hard to detect by palpation only, but the distended duct usually demands exploration. The biliary passages should be opened and all hydatid membrane evacuated with the aid of a soft catheter attached to a suction pump. It is usually possible, as in Cases II and III, to pass the catheter up the right hepatic ducts and to remove cysts from this situation, and probably from the interior of the cyst itself. After the surgeon is convinced that he can obtain no more *débris*, he should drain the duct and search the liver, remembering the favourite subphrenic position and that multiple cysts may introduce the difficulty of detecting which one is at fault. Sometimes the direction taken and the depth attained by the catheter may help in determining its site, while a little 12·5% sodium iodide solution may be used for radiographical study later, particularly if a portable X ray machine is available. If the cyst is accessible, it should be drained as in the preceding cases; if deeply placed, it should be left in the hope that it has been evacuated; if subphrenic, it should be attacked by the usual route, either at the time, if the patient's condition allows, or as soon as the patient has recovered from the effects of his first operation. As a rule the low tension in the cyst and the lessened amount of its contents preclude much risk of the common duct's being blocked again very soon.

2. When, from the evidence of cysts in the stools, from radiography or immunity reactions, the correct diagnosis is made, there are two types of cases to be considered.

(*a*) When the obstruction to the common duct, as evidenced by lessening jaundice and normal stools, has been overcome—and it is advisable whenever possible to wait for this event, because it is not likely that it will become blocked again if the pressure on the cyst is relieved—the offending cyst should be attacked through the ribs by a one-stage or two-stage procedure and drained. It must be remembered that these cysts are practically always infected and that pleural contamination is a real risk, and that this can usually be avoided by making the incision well forward, by producing adhesions by preliminary suture of the diaphragm to the chest wall and packing, or by an anterior approach. The difficulties of localization and the problem of the lax cyst have already been referred to. Following evacuation of the cyst, it is rare in Australian experience for the common duct to remain or to become blocked again. The stools regain their normal colour and the external biliary fistula closes. Sometimes, however, as in Case III, the biliary passages remain blocked by membrane and inspissated bile, and choledochotomy must be performed to clear them. This is carried out in the manner indicated below.

(*b*) When the biliary distension is persistent and of long duration and the patient is obviously ill, perhaps suffering from infective cholangitis, the common duct must be opened and cleared out first as indicated above. The attack on the offending cyst is usually necessary at a later date; but sometimes it seems that practically complete evacuation by means of the catheter and sucker can be carried out, as in Case II. In this case a second operation, with exploration of the upper part of the liver, revealed a deeply placed, collapsed and apparently empty cyst. It is obvious that in these cases a great deal depends on the size of the original cyst and on the judgement of the surgeon.

ACKNOWLEDGEMENTS.

I have to thank Professor Dew for permission to report Cases I, II and III, for help in compiling the article, and particularly for his assistance in the section on treatment. I also wish to thank Mr. B. Edye for permission to report Cases IV and V.

REFERENCES.

(1) Harold R. Dew: "Hydatid Disease", 1928.
(2) F. Dévé: *"L'ouverture des kystes hydatiques du foie dans les voies biliaires", Extrait de la Normandie Médicale*, October, 1932.
(3) B. T. Zwar: "Intrabiliary Rupture of an Hepatic Hydatid Cyst", *The Australian and New Zealand Journal of Surgery*, June, 1931.
(4) Ricardo Finochietto: *"Quistes hidaticos del higado abiertos en las vias biliares", Segundo Congreso Nacional de Medicina*, 1932.
(5) F. Dévé: *"Echinococcose hépatique", Nouveau Traité de Médecine*, Volume XVI, 1928.
(6) Oscar Ivanissevich: *"Litiasis biliar? Quiste hidático? El cólico hepático de origen hidático", Boletin del Instituto de Clínica Quirurgica*, Volume II, 1927.
(7) Geroulanos and Maccas: Quoted by Dévé, *loco citato*.(5)

Reports of Cases.

POLYCYTHÆMIA.

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Case I.

The patient, a male, aged fifty-five years, presented himself on November 25, 1927, complaining of breathlessness, general discomfort, pain in the right hypochondrium, and of his increasing dark bluish-red coloration.

On examination the corneal and retinal vessels were found to be injected; the peripheral arteries were thickened and tortuous, while the systolic blood pressure was 170 and the diastolic 110 millimetres of mercury. There was no evidence of splenic enlargement. Examination of the blood showed the red cells to number 10,600,000 and the white cells 10,000 per cubic millimetre. The hæmoglobin value was 160%. Treatment by phenylhydrazine hydrochloride was instituted.

On February 25, 1928, the red cell count was 4,000,000, the hæmoglobin value was 91%, and the spleen was palpable one finger's breadth below the costal margin. The administration of phenylhydrazine was suspended.

On May 22, 1928, the red cells numbered 8,700,000, the hæmoglobin value was 158%, and the giving of phenylhydrazine was cautiously resumed.

On February 26, 1929, the red cells numbered 6,500,000 per cubic millimetre, the hæmoglobin value was 112%, and the spleen was palpable three inches below the costal margin. The patient was very well and his weight had increased 6·3 kilograms (one stone).

On March 10, 1931, the patient developed bilateral thrombosis in the superficial veins of both legs.

In February, 1932, he was reported to have died suddenly after more than three years of comparative comfort. During the last six months he had experienced one sudden attack of unconsciousness, lasting half an hour, and numerous spontaneous hæmorrhages in various parts of the body, especially in the pharynx and subcutaneously in the neck.

Case II.

The patient was a male, aged sixty-three years. On November 4, 1927, he had dyspnœa, pain round the lower part of the chest, and recent severe epistaxis. His appearance was characteristic, the systolic blood pressure being 208 and the diastolic 150 millimetres of mercury, while the peripheral arteries were tortuous and thickened. The red cells numbered 8,900,000 per cubic millimetre, and the hæmoglobin value was 170%.

On December 6, 1927, treatment by phenylhydrazine was instituted.

On February 5, 1929, the red cells numbered 6,800,000 per cubic millimetre, the hæmoglobin value was 134%, while the systolic blood pressure was 160 and the diastolic 120 millimetres of mercury.

On August 2, 1932, the patient was accidentally killed. Up to this time he had remained in an improved general condition, without complications, on such dosage as maintained a partially restored blood condition.

Case III.

The patient was a male, aged fifty-six years. On August 24, 1934, he reported with slight residual hemiparesis, due to an apparent cerebral thrombosis which had occurred three months previously. For an indefinite number of years preceding this occurrence he had noticed that his face was purplish red, his eyes bloodshot, and he suffered from intermittent giddiness and vague general discomfort.

On examination the radial arteries were found to be thickened and tortuous; the systolic blood pressure was 190 and the diastolic 120 millimetres of mercury; red cells numbered 8,010,000, white cells 14,400 per cubic millimetre, and the hæmoglobin value was 160%.

On September 11, 1934, a course of treatment was instituted with *Liquor Arsenicalis* (British Pharmacopœia), given by mouth, 0·18 cubic centimetre (three minims) three times a day, increased by 0·06 cubic centimetre (one minim) three times a day every third day until a limit of tolerance was reached at 0·72 cubic centimetre (12 minims), whereupon the dose was maintained at 0·3 cubic centimetre (five minims).

On February 19, 1935, the red cells numbered 5,850,000 per cubic millimetre and the hæmoglobin value was 130%; the systolic blood pressure was 158 and the diastolic 100 millimetres of mercury.

On March 27, 1935, the patient was admitted to hospital with left basal pneumonia. The red cells numbered 4,810,000 per cubic millimetre, the white cells 5,600, and the hæmoglobin value was 100%. Arsenical treatment was suspended.

On April 18, 1935, the red cells numbered 5,240,000 per cubic millimetre, and the white cells 11,100; the hæmoglobin value was 100%. The patient was discharged from hospital.

On July 2, 1935, there were 5,900,000 red cells and 12,500 white cells per cubic millimetre; the hæmoglobin value was 120%. The patient's appearance had improved; his weight had increased and there was no disability due to the polycythæmic state. The spleen remained impalpable throughout. The patient was told to resume taking *Liquor Arsenicalis*, 0·3 cubic centimetre (five minims) three times a day.

Comment.

These cases, all of the Geisböck type, are deemed worthy of reporting, to serve as an aid to readier diagnosis and for reviewal of the present methods of treatment. The symptoms resulting from polycythæmia, though manifold, are in no way distinctive, being such as are to be expected from general vascular plethora and an enhanced liability to vascular accidents. They are apt to be ignored when, as is often the case, they are of indefinite nature or attributable to some other cause; especially if, as in a majority of the patients, the blood pressure is raised. But the layman's assumption that every ruddy, bloated countenance connotes alcohol or "blood pressure" should not be accorded our entire acceptance. The classical coloration, which is not purely a matter of capillary plethora, since digital pressure demonstrates actual pigment deposit in the tissues, is occasionally absent. The spleen, as in Case I, may become palpable only in response to a *régime* of treatment that accelerates internal blood destruction. Hence examination of the blood remains the only absolute diagnostic criterion.

Methods of treatment available today are limited to the affording of palliative relief by reducing the number of cells in the circulating blood, either by direct removal (venesection), promoting hæmolysis *in vivo* (with phenylhydrazine or, better, acetylphenylhydrazine) or by depression of blood cell formation (by X ray treatment of bone marrow or by the administration of arsenic).

Though prolonged remissions have been observed, with or without treatment, no method has yet shown itself to admit of ultimate permanent suspension. Nor can we be sure that any of these procedures will not hasten the anæmia or other blood dyscrasia which is so common a terminal event in those who survive intercurrent complications. It is probably wiser in any case to be satisfied with restoration of personal comfort, rather than to insist upon a reduction to a theoretically normal blood picture. Venesection is reserved rather for emergency. It is an uneconomical drain on both cell and fluid elements of the blood and has been shown by an ensuing reticulocytosis (with which, however, all observers are not in agreement) to be provocative of an obviously undesirable marrow stimulation. This last result is proven by independent workers to follow also upon the administration of phenylhydrazine. The procedure with this preparation has been to give an initial course of two to four grammes twice daily in capsules, each containing 0·05 gramme, and to wait about three weeks—hæmoglobinuria continued for this period in Case I after suspension of the drug—before checking the maximum effect by an examination of the blood. On the result of this procedure, and in course of time, according to the personal impressions of an intelligent patient, the need for additional courses or the amount of maintenance dose (usually 0·1 to 0·4 gramme per week) is assessed. Tolerance and degree of response vary with individuals and at different times in the same individual. This is partly accounted for by the greater comparative resistance possessed by younger red cell membranes and so depends upon the relative number of such in the circulation. A period of seeming resistance to the drug may give place to a sudden unexpectedly brisk hæmolysis. Cases have recently come under notice in which very grave anæmia has been provoked by an imperfect knowledge or insufficient care in the use of this drug.

Expense and difficulty of control are somewhat of a bar to the routine use of X ray treatment.

The so-called potassium arsenite treatment,[1] adopted in Case III, was at least equally as effective as the phenylhydrazine. There was no greater production of unpleasant reactions and no anxiety in regard to thrombosis or secondary stimulation of the marrow.

Owing to the clinical contrast presented by the polycythæmic state, ætiological considerations have led to the postulation of a hormonal influence antithetic to that existing in pernicious anæmia. This is strongly supported by demonstration of the fact by Wilkinson and Klein[2] that an extract prepared from the liver of a deceased polycythæmic patient is more potent in the treatment of pernicious anæmia than is one from either calf or normal human liver; and also by a case report of the restoration and control over a lengthy period of a polycythæmic blood picture by repeated gastric lavage alone. While awaiting the possible development of specific therapy along these lines, there exists, therefore, sufficient justification for deleting from the dietary substances now known to contain the anti-anæmic factor or to be productive of the extrinsic factor of Castle.

References.

[1] C. E. Forkner, T. F. McNair Scott and S. C. Wu: "Treatment of Polycythæmia Vera (Erythræmia) with Solution of Potassium Arsenite", *Archives of Internal Medicine*, Volume LI, page 616.

[2] J. F. Wilkinson and L. Klein: "The Hæmopoietic Activity of the Normal and Abnormal Human Liver, with Special Reference to Pernicious Anæmia", *The Quarterly Journal of Medicine*, July, 1934, page 341.

Reviews.

MATERIA MEDICA AND THERAPEUTICS.

HALE-WHITE'S "Materia Medica and Therapeutics" is so well and favourably known to medical practitioners and students that it has now reached its twenty-second edition.[1] However, with the elimination of the subject of *materia medica* and therapeutics as a separate entity from medical curricula, possibly the demand for this most excellent work will diminish. The new edition contains many trade preparations of undoubted utility and other unofficial preparations. Amongst these a few may be indicated. "Atebrin" is a synthetic dye, an acridine preparation. In malaria it is best used with quinine. It may colour the skin yellow and cause abdominal pain. "Cardiazol" and "Coramine" have a camphor-like action and are of benefit in cardiac and respiratory failure, especially in narcotic poisoning. "Dilaudide" is a morphine derivative three times as powerful as morphine. "Dicodide" is a codeine derivative similar in action to "Dilaudide", but less toxic. Although addiction is stated to be less than with morphine, both should be held in suspicion pending further knowledge. "Evipan" and the other barbiturates receive attention, but, curiously, "Somnifaine" is not mentioned. "Novutox", a cocaine substitute, is considered, but no reference is made to any possibility of addiction. The complex organic mercury compounds "Neptal" and "Salyrgan", as diuretics, are mentioned. "Icoral" is a synthetic preparation of two bases. It raises the blood pressure and stimulates respiration, being of value in collapse and hypnotic poisoning. Pentnucleotide causes a polymorphonuclear leucocytosis and has proved beneficial in agranulocytic angina, although it at times produces alarming symptoms. The anterior pituitary hormone is correctly stated to contain probably two hormones. Choline, which is allied to muscarine, and acetyl-chlorine, which acts as a natural hormone and is liberated at the nerve-endings when the parasympathetic is stimulated, are both stated to cause slowing of the heart and arteriolar dilatation. In the part dealing with the action of drugs on the nervous system the relationship between nerve impulse transmission and the production of chemical bodies is briefly explained. In this connexion acetyl-chlorine has already been mentioned. Stimulation of the sympathetic gives rise to an adrenaline-like body, "sympathin", which probably includes two substances. In regard to the vitamins, it is stated that vitamin *A* does not occur in cod liver oil, when it was obviously intended to indicate that carotene does not so occur. Again, under vitamin *E* we are told that it occurs in "wheat, germ oil", which should read "wheat-germ oil".

We are informed of cineole under eucalyptus oil, but it appears incorrectly as cineol, under cajuput oil. It is a pity that the recommendations of the last Pharmacopœia regarding the writing of prescriptions have not been followed. However, the book amply preserves the excellence of former editions. One can recommend especially the rewritten chapter on digitalis.

[1] "Materia Medica, Pharmacy, Pharmacology and Therapeutics", by W. Hale-White, K.B.E., M.D., LL.D.; Twenty-Second Edition, revised by A. H. Douthwaite, M.D., F.R.C.P.; 1935. London: J. and A. Churchill. Crown 8vo., pp. 552. Price: 10s. 6d. net.

FORENSIC MEDICINE.

A NEW text book of forensic medicine, by Dr. Douglas Kerr, of Edinburgh, has been written with the special object of teaching forensic medicine to students and also as a modern guide for the general practitioner.[1] The book as a consequence has a limited compass, but nevertheless covers the subject very fully. The book follows the usual well known lines and the text is very readable.

The introduction deals with the General Medical Council and professional ethics and secrecy, and the book proper commences with the legal procedure to be followed in cases of death in England and Scotland. In Australia the procedure is somewhat different, though the same in principle.

The method described of making medico-legal *post mortem* examinations should be useful to practitioners who are not in the habit of making such examinations, but could have been dealt with in more detail. The procedure to be followed in cases of anæsthetic deaths is a welcome inclusion and is one not often found in text books.

The book then deals with identification. Notable omissions here are references to the centres of ossification and a more detailed description of the bones. Following this is an account of the changes that occur after death, followed by a short chapter on death and sudden death.

The chapter on wounds and wounding is rather meagre. One would appreciate their treatment more fully at the expense of remarks on malpraxis and consent.

For a book of its size, the chapter on the examination of blood and hairs is well and fully done.

The author has handled the chapters on heat, cold and starvation and on asphyxia excellently.

Pregnancy, abortion, infanticide and sexual offences are adequately dealt with.

Too much space is given to insane states, criminal responsibility and legal aspects of insanity in a book of this size and nature. We feel that the fuller treatment of these subjects is better left to the text books on insanity and the larger treatises on medical jurisprudence. A short description of the Binet-Simon tests as given seems to be quite unnecessary.

Toxicology is very fully treated and a pleasing inclusion is the poisons schedule of 1935. The inclusion of many proprietary preparations, especially the barbiturates, will be appreciated by the general practitioner. No mention, however, is made of the lumbar puncture method of treatment in cases of barbiturate poisoning. Food poisoning is treated rather too briefly, as is also snake-bite. No mention is made of the use of antivenene.

Poisoning by alcohol and drunkenness are discussed very completely for a book of this size and the tests and conclusions arrived at should be of great help to those who are called in by the police to decide on this question. The illustrations are clear and numerous.

Altogether, in spite of several omissions, the subject has been excellently treated by the author and the book is one which can be thoroughly recommended both for the general practitioner and the student.

[1] "Forensic Medicine: A Text-Book for Students and a Guide for the Practitioner", by D. Kerr, M.D., F.R.C.P.E., D.P.H.; 1935. London: A. and C. Black, Limited. Demy 8vo., pp. 321, with illustrations. Price: 15s. net.

The Medical Journal of Australia

SATURDAY, NOVEMBER 23, 1935.

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DOCTORS' HOBBIES.

EXHIBITIONS of the hobbies of doctors always arouse not only admiration, but surprise. This is hardly fair, for it is perhaps a little inconsistent that members of a profession that is, or should be, an intellectual one and that makes wide contacts with the world of men should be at all surprised at the collateral activities of their brethren. It is a truism that only the busiest people can find spare time, but we may be allowed to express genuine pleasure, if not astonishment, that doctors can in their odd hours of relaxation attain high degrees of skill in arts and crafts more or less remote from their calling. No one who saw the exhibition of hobbies displayed during the recent British Medical Association meeting in Melbourne could fail to be impressed by its range and quality, but it must be remembered that even this collection is by no means representative of the sum of what we might call medical non-medical activities.

There is in this subject of hobbies a matter of great interest and importance. At frequent intervals we hear laments that the standard of culture and of general education of doctors is falling, and it is pointed out that the growth of medicine and surgery continues at so rapid a rate that almost the whole of the energy of any thoughtful and ambitious practitioner is absorbed in keeping himself up to date. The latter at least is true; there were fourteen sections in the Melbourne meeting alone, and every general practitioner must be able to acquire and to retain at least a modicum of knowledge of each of the subjects that these sections cover. Therefore it is perhaps timely to point out the danger of isolation from the outer world and of a too complete absorption in the work of every day. Clifford Allbutt, himself an admirable example of the learned and polished doctor, declared that every medical man should have both a vocation and an avocation: a vocation, the life work to which he was called by choice and circumstance; and an avocation, an occupation which called him away from his daily labours. No doctor should ever become like the great scientist who, after a life spent in specialized work, grieved to find that he had lost his taste for the arts; or like Robert Walpole, who roved desperately around his splendid library exclaiming: "It is all in vain; I cannot read." Yet do we not warn medical students that they are entering the service of a stern mistress who will allow no rivals, no diversity of interests? Such sentiments fall even from distinguished lips upon the ears of those young and as yet enthusiastic neophytes who still have that blessed capacity of being interested in most things. Let us cultivate our avocations. It is not enough to have "some suburb palace, parked about and gated grandly", nor yet a "Mayfair residence, fit to receive and entertain"; more desirable, as Browning avers, is the "hole i' the wall", the "cupboard suited to his mind", where the doctor forgets his "shop" and pursues his hobby. It is perhaps natural that the devotees of a calling which is in part a science and in part an art should turn aside to handicrafts and to the arts of painting, literature, music and the like; but whatever the doctor's "avocation", let us encourage it. Browning again has the truth:

> I want to know a butcher paints,
> A baker rhymes for his pursuit,
> Candlestick-maker much acquaints
> His soul with song, or haply mute,
> Blows out his brains upon the flute.

Current Comment.

ANÆMIA IN THE CHRONIC RHEUMATIC DISEASES.

ANÆMIA has been known and recognized as part of the syndrome of juvenile rheumatism during the whole of the present century. The common association of anæmia with rheumatoid arthritis led to the successful use of blood transfusion as part of the treatment for the latter condition. Pallor has been authoritatively indicated as pointing to a diagnosis of infective arthritis in the presence of chronic arthropathy. Such anæmia is in general of the hypochromic, chlorotic type.

Douglas H. Collins has investigated the effect of massive doses of iron on blood deficiency in arthritis with disappointing results.[1] His chief findings relate to 196 patients attending the Harrogate Royal Bath Hospital. A full hæmatological examination was performed upon 110 patients. Collins found that 58% of the men and 77·5% of the women had hæmoglobin values below the limits of normality. Of such patients, 41·9% suffered from atrophic (infective or rheumatoid) arthritis. No example of hyperchromic anæmia was encountered, and in general the red count was much less affected than the hæmoglobin. In order to determine whether anæmia is commoner in any particular clinical type of rheumatism, the series of patients investigated was carefully classified into three main groups: those suffering from atrophic arthritis, those with osteoarthritis, and those with non-articular rheumatism. Two important findings were recorded: first, that anæmia is far more common among female than among male patients, six times as many females as males having hæmoglobin values below 12 grammes *per centum*; secondly, the severer grades of anæmia occur only in atrophic arthritis. Fractional gastric analysis, after administration of the oatmeal meal and without histamine stimulation, was performed upon 100 patients. While the females suffering from atrophic arthritis provided most of the examples of anacidity encountered, it was clear that achylia was not a constant feature of the anæmia accompanying chronic arthritis. It was also apparent that severe anæmia might develop in this disease even in the presence of a normal gastric secretion. Collins remarks that it is surprising how often the blood picture in such cases will be found to be normal or nearly normal. The age of the patient appeared to have no influence upon the anæmia, while long duration of the joint disease coincided with the more pronounced degrees of blood deficiency. In other words, the anæmia was secondary to the arthritis.

It has been the custom in the treatment of rheumatoid arthritis to make use of diets low in carbohydrate, which in some cases may interfere with the iron supplies of the organism. Collins attended to this point and found only two instances in which it might have operated as a cause of microcytic anæmia. Both these patients responded satisfactorily to the oral administration of iron.

Collins's work constitutes a valuable piece of practical research, and is the type of investigation now being undertaken by the full-time research workers at the English spas. Much that is traditional rather than rational, many local fads and therapeutic techniques will ultimately be reviewed from a more modern laboratory angle. It may be confidently anticipated that work of this kind will throw light on much of the obscurity and even humbug surrounding this field of medicine.

[1] *The Lancet*, September 7, 1935.

ICTERUS NEONATORUM.

MANY conditions may result in jaundice in the new-born. Chronic hæmolytic jaundice may be congenital and, at the outset, may resemble physiological icterus in its manifestations. Congenital obliteration of the bile ducts leads to intense obstructive jaundice, visible at birth. Familial jaundice in some, but not all, cases is hereditary. It may occur in several generations and in different members of each generation. R. Hutchison states that an unaffected member does not transmit the disease. The grave familial form of jaundice seems less likely to attack first and second than later children. It resembles physiological jaundice in its autopsy findings, particularly in the selective staining of the lenticular and other cerebral nuclei and in the early onset of febrile jaundice, with bile in the fæces, but, as pointed out by F. Langmead, the urine may contain no bile pigment. Congenital anæmia accompanies fatal familial jaundice. The death rate of familial jaundice of the new-born reaches 80%, but has been conspicuously reduced by injections of maternal blood serum. The disorder called *icterus neonatorum* is a different entity. It is not certain whether it is primarily hepatogenous or hæmatogenous in origin. E. F. Murray, reviewing 337 infants, found that the onset of jaundice was mostly on the first day and was rarely delayed until the third or fourth. The duration varied greatly, but was usually less than a week. The earlier the onset, the severer was the icterus. The small child had the greater tendency to jaundice. Murray stated that the amount of blood obtained by the infant from the placental circulation before ligature of the cord affected the incidence of jaundice—a small amount predisposed to the condition, while a normal or excessive quantity was antagonistic. Murray suggested that when early separation was necessary, the cord should be ligated as far from the child as possible, so that it could aspirate the blood in the cord. This applies particularly to asphyxiated infants who have a low blood count. The Van den Bergh test shows that in *icterus neonatorum* the bilirubin in the blood serum is the same as that in hæmolytic jaundice, differing from that of obstructive jaundice.

N. Book points out that visible jaundice in the new-born has been variously estimated at from 60% to 90%.[1] He considers that it generally begins on the second or third day and that the infant generally suffers no immediate ill-effects. He enumerates the various theories as to its causation, but considers that the ætiological factors most worthy of consideration are polycythæmia, corpuscular fragility, and the state of the liver and the spleen. Pronounced polycythæmia is present at birth, but gradually subsides. This polycythæmia is compensatory. As a respiratory organ the placenta is inadequate and the fœtus lives in a state of chronic oxygen starvation. The hæmoglobin percentage is elevated at birth and drops synchronously with the fall in the red cell count. The red cells at birth have fragile envelopes and readily break down. Normal stability is regained about the tenth day. In the first few days of life the liver and spleen are still hæmatopoietic organs and are not sufficiently mature to dispose properly of the broken-down red cells and hæmoglobin. Book considers that these factors alone are enough to produce icterus and that they probably do so. The majority of cases of jaundice in infants belong to the group of simple physiological jaundice. Other types are differentiated, particularly *icterus gravis*. This has been estimated to occur once in 340 births. Anæmia is a prominent feature and the mortality is very high, some patients dying in three or four days. Necrosis of the liver cells and early fibrosis have been described. Cerebral staining with bile, particularly of the medullary nuclei (*Kernikterus*) is often present. Some attribute to this condition the spasticity, athetosis and mental defects in recovered patients. In *icterus gravis* immature red cells occur in the blood, indicating intense regenerative activity. In infants who have died, foci of blood-forming tissue have been found all over the body—a condition of erythroblastosis. They signify a reversion to the embryonic state to combat the hæmolytic process, the bone marrow being inadequate. Whether simple physiological jaundice can progress to *icterus gravis* is a moot point. Some authorities hold that it can. Cerebral staining with bile has been found in the simple physiological group. Book considered that if physiological jaundice could become pathological, lowering the incidence and severity of jaundice in the new-born should reduce the occurrence of *icterus gravis*.

Book's speculations are at variance with those of Murray. Book compared the immediate tying of the cord with the usual method of waiting till all pulsations had ceased. He examined the records of 200 cases in each class and was convinced that, when the cord was tied immediately, there was a reduction in the percentage of jaundiced infants and the degree of jaundice was decreased. His explanation of the benefit is that the cord and placenta contain about 100 cubic centimetres of blood and that the additional amount of blood the infant will receive when the cord is tied only after pulsations have ceased is about 40 to 50 cubic centimetres. In a seven pound (3,175 gramme) baby the blood volume is one-twentieth of the body weight, that is, 150 cubic centimetres. The extra 40 to 50 cubic centimetres received by the infant whose cord is tied late will overload the circulation by over 25%. Such overloading occurs in blood already embarrassed by polycythæmia and increased fragility of the red cells. The condition, abnormal to begin with, would tend to be exaggerated. Excluding this extra amount of blood would be beneficial. Book's speculations are interesting, but his findings are far from being established. He suggests further work on the same lines on a larger series of cases to corroborate or refute the findings.

[1] *The Canadian Medical Association Journal*, September, 1935.

ABSCESS OF THE LUNG AND DENTAL OPERATIONS.

IN a recent communication[1] Leo Stern is at considerable pains to show that pulmonary abscess following dental operations is not so rare an occurrence as is generally thought. He states that such an abscess is usually supposed to follow aspiration of a tooth following extraction. He has investigated a series of seventy lung abscesses and finds that in twelve instances the formation of an abscess followed dental operations; in no case was there any clinical or radiological evidence of aspiration of a tooth. The question is one of great importance, though from the preventive point of view it concerns the dental surgeon rather than the medical practitioner. Stern points out that particulate matter in the mouth may easily be aspirated into the lungs and that the bacteriological findings in abscess of the lung after dental operations are similar to the flora associated with the so-called pyorrhœa pocket. Aspiration is most likely to occur with loss of the pharyngeal reflexes. Local anæsthesia was employed in eight of the twelve cases reported by Stern, nitrous oxide was used in three, and in one no anæsthetic was used. In five cases teeth were removed from the posterior part of the maxilla, where the customary procedure is to block the anterior palatine nerve which affects the reflex mechanism of a portion of the soft palate. Stern makes the bald statement that at the present time no attempt at a scrupulous hygiene is made before a tooth is extracted. There is justification for his contention that the preparation for extraction of a tooth should include high pressure irrigation of the interdental spaces, the use of dental floss, and especially the painstaking removal of tartar. The gingival margin should be cauterized by heat after anæsthesia has been induced; the patient should be prone during extraction; and extractions should, he thinks, be performed in the morning, so that time is allowed for complete control of hæmorrhage before the patient ordinarily sleeps. Medical practitioners would be wise to discountenance the use of hypnotics immediately after tooth extraction and to insist on the routine use of a peroxide of hydrogen mouth wash.

[1] *The Journal of Thoracic Surgery*, August, 1935.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Chorea and Rheumatic Fever.

T. DUCKETT JONES AND EDWARD F. BLAND (*The Journal of the American Medical Association*, August 24, 1935) analyse the prognostic significance of chorea as a manifestation of rheumatic fever. They believe that the association between true Sydenham's chorea and rheumatic fever is too close to warrant a separation of the symptom complex from the syndrome of rheumatic fever. However, they consider chorea to be a mild manifestation of rheumatic fever and one in itself not especially conducive to the development of rheumatic heart disease. They made their observations in a group consisting of 482 patients with chorea and 518 patients presenting manifestations of rheumatic fever other than chorea. The incidence of rheumatic heart disease was 86% in the group of patients with rheumatic fever without chorea and 54% in the patients with chorea. Furthermore, when the patients with chorea were further analysed into those with chorea and other manifestations of rheumatic fever, and those with chorea but no other manifestations of rheumatic fever (pure chorea), the incidence of rheumatic heart disease was found to be 73% in the former and 3% in the latter group. The manifestations of rheumatic fever other than chorea would thus appear to be important factors in the development of rheumatic heart disease. The number of attacks of chorea does not appear to influence significantly the development of rheumatic heart disease. The severe accompaniments of rheumatic fever occur twice as frequently in patients having rheumatic fever as in those having chorea in addition to rheumatic fever. Finally, the death rate is seen to increase noticeably in the groups of patients exhibiting more evidence of rheumatic fever than chorea. The authors conclude that the prognostic significance of chorea as a manifestation of rheumatic fever has evidently been exaggerated in the past.

Undescended Testes.

LE ROY BROOKS (*Archives of Pediatrics*, July, 1935) discusses the treatment of undescended testes. He points out that the usual location of an undescended testis is within the inguinal canal, and that it is common knowledge that in certain cases the testis will descend spontaneously before or at about the time of puberty. The administration of "Antuitrin-S" seems to have been shown to cause early descent in some cases. It is equally well known by surgeons that the usual finding at operation is a testis firmly fixed to the tissue of the inguinal canal by the gubernaculum. Further, research work has shown that the testis should be placed within the scrotum if normal development in size and composition is to be expected. It is now agreed by most authors that the sooner the testis is placed within the scrotum, the better. Early operation also removes the undesirable psychological factor present in growing boys with undescended testes. The author's experience leads him to prefer to operate upon these children between two and three years of age. The operation for the condition is described. Reference is made to the perplexing problem as to whether any given case belongs to the endocrine group, in which descent will occur spontaneously or with the help of "Antuitrin-S", or to the mechanical group, in which there is fixation of the testicle by fibrous tissue within the inguinal canal. The motility of the palpable testis is of some value in this respect. Since the best results can be secured by an early operation, it would seem desirable to try the use of "Antuitrin-S" at from two to two and a half years of age, and if this does not cause descent, operation would be indicated. The author asks for information as to what untoward effects, if any, are to be expected from the use of "Antuitrin-S", and how often, how long, and what dosage is necessary to determine whether or not success with the extract is going to be obtained.

The Diagnosis of Congenital Syphilis.

A. H. PARMELEE AND J. L. HALPERN (*The Journal of the American Medical Association*, August 24, 1935) draw further attention to the overwhelming significance of osseous lesions as revealed by X rays in the diagnosis of congenital syphilis. They studied a series of 104 consecutive infants born of mothers known to have syphilis. Clinical, serological, and X ray examinations were made during the first few days of life, and at the age of six weeks, three months, six months, and one year. During the new-born period there was suggestive clinical evidence of infection in 19% of the cases, serological evidence in 23%, and X ray evidence in 44%; at six weeks the figures were 30%, 8%, and 61%; at three months they were 25%, 8%, and 63%; and at six months they were 34%, 13%, and 47%. The authors' observations indicate that too much reliance on the serum reactions during the new-born period may lead to errors in diagnosis. In regard to the radiological appearance of the bones, the picture is predominantly that of an osteochondritis. The earliest manifestation is the thickened epiphyseal line with a narrow zone of rarefaction immediately shaftward in the metaphysis. Periostitis is almost never present in the new-born.

Observations on Rheumatism in Children.

T. WARWICK PRESTON (*British Journal of Children's Diseases*, January-March, 1935) reviews the work of the past four years at the Elizabeth Bullock, Putney and Downham rheumatism supervisory centres. Particular attention is paid to the evaluation of "growing pains" as a manifestation of rheumatism in children. The author concludes that pains of this type occasionally prove to be an insidious form of rheumatism which may attack the heart, but that many pains which may appear to be rheumatic are caused by minor orthopædic conditions and general debility. It is suggested that some children who continue to complain of aching limbs over a long period, for which there is no adequate explanation, and who on the evidence available are unlikely to develop carditis, may be suffering from a form of septic myofibrositis analogous to that of adult life. The fact that many of these children are cured by removal of a septic focus supports this view. "Insidious carditis" is usually the result of a definite subacute attack of rheumatism, which may be so mild and transitory as to escape notice. It is not usual for children so affected to give a long history of growing pains. Further, growing pains are uncommon in children definitely known to have had acute rheumatism. Among the children with serious rheumatic carditis the general physique is remarkably good and complaints of all kinds are rare. Nevertheless the incidence of relapse is high in spite of every care. These children should be kept under frequent medical supervision, however well they may appear and however free from symptoms.

Whooping Cough.

HUGH C. THOMPSON, JUNIOR (*Archives of Pediatrics*, August, 1935) has investigated the prophylactic and therapeutic effect of pertussis vaccine made by the New York State Department of Health, and also the therapeutic effect of elixir of gold tribromide. The investigation was carried out during an epidemic of twenty-six cases of pertussis occurring in an infants' home, all the patients being less than two years old. Every other child was given three doses of the pertussis vaccine and comparison with control cases showed no appreciable prophylactic or therapeutic effect. Elixir of gold tribromide was given during the spasmodic stage of the disease to eight infants who had not received vaccine. In this group there was less loss of weight than in the other children, and some of the patients seemed to cough less during the period in which the elixir was being administered. The author concludes that neither of these therapeutic agents gave results which could be described as satisfactory or encouraging.

ORTHOPÆDIC SURGERY.

Recurrent Dislocation of the Patella.

F. OBER (*The Journal of Bone and Joint Surgery*, July, 1935) describes his operative treatment for recurrent dislocation of the patella. He has

noted in the past that the Goldthwait operation sometimes fails to hold the patella in its normal position. In such a case he found the patella situated beneath the ilio-tibial band, to which it was connected by another band which had to be divided before reduction was maintained. The patella is usually dislocated laterally owing to the fact that it is placed at a higher level above the knee joint than is normal and so does not have the support of the lateral condyle of the femur. The procedure he describes is carried out through a 15·0 or 17·5 centimetre (six or seven inch) incision extending upwards along the lateral aspect of the thigh, exposing the ilio-tibial band, from which a strip 15·0 centimetres (six inches) long and 1·25 centimetres (one-half inch) in width is dissected, leaving the lower end attached to the patella. The superficial and deep bands from the lateral aspect of the patella are divided, care being taken to avoid entering the knee joint. Through another incision over the medial border of the tibia a tunnel 1·25 centimetres (one-half inch) long is made through this bone and the flap of the ilio-tibial band is passed down through a tunnel beneath the aponeurosis of the patella and is brought through the tunnel in the tibia, from within outward, drawn as tightly as possible, and sutured to the surrounding structure. The result of this operation is to provide an extra patellar tendon, which prevents redislocation.

The Morbid Anatomy of Caries of the Thoracic Spine in Relation to Treatment.

H. J. SEDDON (*The Lancet*, August 17, 1935) classifies the vertebral manifestations of tuberculosis into: (i) so-called tuberculous epiphysitis, which is really a metaphysitis, seeing that the lesion is found on the upper or lower surface of the body; (ii) diffuse osteomyelitis, which is indicated by an increase in the density of the vertebral body, indicating a necrosis of bone with deposits of lime salts, which may be confused with the signs of healing; and (iii) the so-called periosteal lesion, which cannot be diagnosed until erosion of the bodies has occurred or until the cord has been compressed by an abscess beneath the anterior or posterior spinal ligament. Bone destruction occurs as a result either of tuberculous ulceration or mechanical ulceration, or both. Mechanical ulceration is due to friction resulting from movement between inflamed and softened joint surfaces, which movement is exaggerated in the dorsal region by the respiratory movements of the thorax. Destruction of the vertebral body may also be due to erosion of several bodies from the aneurysm-like pressure of a thoracic abscess confined closely to the vertebral body by the strong anterior spinal ligament. In such cases there is a development of secondary foci above or below the original lesion. Angulation of the spine invariably follows the destruction of a number of vertebræ and may continue for years after the lesion has healed. It cannot be prevented by any form of external support or even by a bone graft in children. The segment of the spine above the angulation may travel anteriorly or posteriorly, the former being the characteristic deformity in severe high thoracic caries, and the latter in extensive lesions of the lower thoracic region. Solid fusion is often found in the spine showing posterior luxation, in which cases the prognosis as regards deformity is better than in those with anterior luxation. The treatment of these conditions again depends on the universal recognition of the value of rest, but this principle, to be of value, must be applied with some very definite fundamental methods. Immobilization combined with powerful longitudinal traction, although it would produce rest, is impracticable for any length of time, yet the same effect may be obtained without difficulty by rest of the spine in hyperextension, the aim of treatment being to maintain a distinct gap between the diseased surfaces and thus to eliminate the mechanical ulceration referred to. By this method bone destruction is reduced to a minimum and accidents resulting from flexion of the spine during the active stage of the disease may all be prevented. Compensatory overgrowth appears to occur only in such cases, and is a particularly valuable result in children. The hyperextension should be maintained until radiography demonstrates a cessation of bone destruction, recalcification of the involved vertebræ and improvement in the definition of the bone where erosion has been taking place. This may take two or three years, and only then may the vertebræ be allowed to fall together and fuse. Compensatory lordoses subsequently develop above and below the lesion, but where bone destruction has been marked, short curves of this nature may be adequately produced by the use of thick pads of sponge rubber immediately above and below the kyphosis while the patient is resting on the posterior frame. Spinal fusion for active disease in children has been tried and found wanting, but there is still apparently a field for its use in rendering permanent the beneficial results of conservative treatment.

Treatment of Giant Cell Tumours of the Long Bones.

C. F. GESCHICKTER (*The Journal of Bone and Joint Surgery*, July, 1935) compares the indications for surgical interference with those for irradiation in the treatment of giant cell tumours of the long bones. In the past decade the tendency has been to refrain from radical surgical interference in favour of curettage and irradiation, but the point has been reached where a middle course can frequently be adopted. Irradiation is preferable in the following group of giant cell tumours: (i) A bone cyst on the shaft side of the epiphyseal line, with a short duration of symptoms. The irradiation does not interfere with the growth of epiphysis. (ii) A giant cell tumour in elderly adults which is seen relatively late and when there is pronounced destruction of bone. (iii) A tumour located in the spine, particularly in the lumbar or cervical region or in the skull. Surgical interference is indicated in preference to irradiation in such bones as the ulna, fibula, and, more rarely, a rib, where the bone is not essential to function; also in patients in middle life in whom the function of the limb is vital to their occupation and livelihood, and in those patients having a recurrence after previous treatment. Accuracy in diagnosis is essential and the author emphasizes the possibility of the radiological findings not being entirely diagnostic. In a follow-up of 200 cases treated by various methods, the percentage of successful results was highest when resection had been performed.

Dislocation of the Cervical Spine.

T. P. BROOKES (*The Journal of the American Medical Association*, March 16, 1935) reviews a series of sixty-five cases of traumatic dislocation of the neck occurring in his own practice during the last six years, and emphasizes the possibility of this condition occurring in coexistence with systemic conditions, such as the prodromal stage of an acute illness or various inflammatory conditions in the neck, which mask the dislocation previously produced. The symptoms differ from those of toxic torticollis in that in the latter. movement in any direction is painful and constant, and the head is rotated and held down on one side by a spastic shortened sterno-mastoid muscle, and the deformity becomes corrected as the condition subsides, whereas in the former condition the shortened sterno-mastoid muscle is soft and flaccid, while the corresponding muscle on the other side is taut and prominent, and efforts to rotate the head in the direction of correction are stopped by a sense of mechanical block. Skiagrams are important as an aid to diagnosis, but inability to demonstrate the dislocation by this method should not settle the diagnosis, as subluxations cannot always be shown conclusively on the films, particularly in the region of the atlas and axis. The author uses the Walton technique for unilateral simple dislocations, and the Taylor technique for bilateral dislocations and those associated with fracture. He emphasizes the need for repetition of this technique in many instances until reduction is maintained, and considers that open reduction in these conditions is of questionable value.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal North Shore Hospital of Sydney on June 20, 1935. The meeting took the form of a series of demonstrations by the members of the honorary staff.

Pulmonary Tuberculosis Treated with Tuberculin.

DR. F. GUY GRIFFITHS showed a male patient, aged forty years, who had been admitted to the Royal North Shore Hospital of Sydney on April 4, 1933, complaining of blood-spitting. Five weeks earlier he had spat a cupful of blood, but continued at work, and a week before admission he had coughed up half a cupful, and since then he had night sweats and malaise and his sputum was blood-stained. For some months he had had greenish-yellow expectoration, but thought it due to "cigarette cough".

On his admission to hospital his pulse frequency was 100, his temperature was 39·2° C. (102·6° F.), and respiratory frequency 28. The left side of the chest was somewhat flattened, and tubular breathing and râles were audible over the left upper lobe and medium crepitations towards the left base; his cough and voice were hoarse; tubercle bacilli were found in his thick, yellow sputum, and the rate of sedimentation of his blood corpuscles was 13. The skiagram, taken in bed, showed a tuberculous lesion of the left apex and pleural adhesions to the left diaphragmatic arch. The patient was propped up in bed and treated with fresh air, light (mostly fluid) diet, aperients, rare doses of morphine cough linctus, and a calcium chloride mixture, 1·0 gramme (15 grains) per dose. As the fever hardly subsided in the first week he was given 0·09 gramme (one and a half grains) of pyramidon in 2·0 cubic centimetres (half a drachm) of aromatic spirits of ammonia and 60·0 cubic centimetres (two ounces) of water every four hours, and by May 10 his temperature had become normal, and injections of albumose-free tuberculin ("T.A.F.") were commenced. Thereafter his recovery was uninterrupted. By May 27 he had no sputum and his strength increased. On June 6 a skiagram showed marked increase of mottling in the left lung, thought to be indicative of an old tuberculous infection, and increase of lung markings of the right lung, suggesting more recent spread of infection. On July 1 the patient was still better and stronger, had no cough or sputum, and his weight had increased to 63 kilograms (ten stone), "heavier than he had been for years".

Dr. Griffiths explained that the first dose of "T.A.F." on May 10 was 0·00002 cubic centimetre, the subsequent doses 0·00004, 0·00008, 0·00012 *et cetera* at intervals of three or four days up to 0·0024—120 times the original—on June 26. Treatment was carried on in the out-patient department and the patient continued to improve; he took daily walks of increasing length, till in September he weighed 69·3 kilograms (eleven stone) and walked six miles and sometimes more, and had reached a dose of 0·02 cubic centimetre of "T.A.F."; the tuberculin was continued once a week up to 0·29 cubic centimetre at Christmas time, and then once a fortnight.

From March to September, 1934, the patient had received no tuberculin, but he then "caught flu" during an epidemic of coryza and bronchitis, and was treated with bovine tuberculin ("P.T."), increasing from 0·002 cubic centimetre on September 13 to 0·15 cubic centimetre on December 6, 1933.

The hæmoptysis did not recur. At the time of the meeting the patient was very well, weighing 67 kilograms (ten stone nine pounds), and had escaped "colds" prevalent in his home and workshop, and had neither cough nor sputum. A skiagram at the end of May, 1935, showed that the condition was much improved since the previous examination. There was still, of course, much pulmonary fibrosis, and it was too early to consider the disease arrested, though the fibrosis was curative. The patient had not been away from Sydney for two years.

Dr. Griffiths's second patient was a woman, aged forty-two years, who had been admitted to the Royal North Shore Hospital of Sydney on August 20, 1934, complaining of hæmoptysis for three days. In the winter of 1933 she had had a "cold" and cough for five months, but recovered fully and continued well and strong till early in August, 1934, she had a similar cold and then coughed blood.

On admission to hospital she was thin and ill, her temperature was 38·3° C. (101° F.), her pulse rate 96 and her respirations numbered 27. Her chest was shrunken; tubular breath sounds were present, with râles and pectoriloquy in the upper part of the left lung and bronchial breath sounds elsewhere (right and left). The sputum was purulent and contained tubercle bacilli; the skiagram showed advanced tuberculosis of both lungs, especially in the upper lobes and more on the left than on the right side; the blood sedimentation rate was 20·5; the urine was albuminous.

The patient's temperature oscillated from 36·7° C. (98° F.) in the morning to 38·9° or 39·4° C. (102° or 103° F.) and occasionally 40° C. (104° F.) in the afternoon, and her pulse rate from 100 to 120. Hæmoptysis of two or three ounces recurred thrice on the day of admission and then less often, necessitating hypodermic injections of morphine on five occasions and a morphine linctus. She was given also one gramme (15 grains) of calcium chloride in a mixture thrice daily, and aperient doses of sulphate of magnesia, tepid liquids, especially eggs and milk, and was left semi-recumbent in bed.

By September 24 her temperature had been normal for four days and she weighed 47 kilograms (seven stone six and a half pounds); she then had her first hypodermic injection of albumose-free tuberculin ("T.A.F."), 0·00002 cubic centimetre, and at intervals of three or four days 0·00004, 0·00008, 0·00012, up to 0·005 cubic centimetre (250 times the first dose) on November 17, 1934. Her expectoration ceased, her weight increased to 49·3 kilograms (seven stone twelve pounds), and when she was discharged from hospital on November 23 she walked freely and felt well and strong.

From September she had taken a mixture of tincture of *nux vomica* and calcium hypophosphite instead of the calcium chloride. She still lived in the northern suburbs of Sydney, and her treatment had been continued as an out-patient up to "T.A.F." 0·4 cubic centimetre (twenty thousand times the first dose) and she still did well. Her weight was 55·5 kilograms (eight stone seven pounds), and the honorary radiographer reported that there were still signs of extensive bilateral tuberculosis of submiliary type.

Recurring Pneumonia.

Dr. Griffiths also showed a boy, aged six years, who was admitted to hospital in February, 1933, on the fourth day of an attack of acute bronchopneumonia. He was treated in a knitted woollen undershirt with an alkaline gentian and *nux vomica* mixture and made a quick and apparently complete recovery.

At the end of August he was admitted with a second and milder attack. Possible sources were sought, but none was found; the skiagram showed "increased lung markings in the hilar regions suggesting chronic bronchitis or possibly tuberculosis", but his sputum was examined on three or four days and no bacilli were found, and he gave no slightest sign of reaction to a hypodermic injection of albumose-free tuberculin, 0·00025, 0·0015 and 0·003 cubic centimetre, at intervals of two days.

In April, 1934, he had a third attack, this time a sudden, very severe lobar pneumonia at the left base. There were still no tubercle bacilli in the sputum, and the skiagram of the lungs showed an area of unresolved pneumonia, but he had pus in the nares and both antra, and the ethmoids were semi-opaque to X rays.

Dr. Clowes had recommended operation, and Dr. Bettington had performed right and left nasal antrostomy. The patient recovered rapidly and again left hospital apparently well. He had since had a mild recurrence of his antral disease. Dr. Griffiths said that if this pro-

gressed, his bronchitis might become chronic or go on to bronchiectasis.

Dr. Griffiths also showed a child, aged one year and ten months, who had been admitted to hospital on April 10, 1935, after having had convulsions. The child's temperature was 40° C. (104° F.), the pulse rate was 172, and the respiratory rate 56 in the minute. No definite dulness was present on the right side of the chest, but bronchial breathing was audible on the right side of the chest posteriorly; rhonchi were present on the right side. A diagnosis of pneumonia was made. Recovery followed a definite, clear-cut crisis. When the child was discharged from hospital on April 23, 1935, the respiratory rate was 25 in the minute.

The child was readmitted to hospital on May 22, 1935, with a history of constant distressing cough and of anorexia for three days. The temperature was 39·4° C. (103° F.), the pulse rate was 170 and the respiratory rate 70 to the minute. Grunting respirations and a short harsh cough were present. Examination of the respiratory system revealed an impaired percussion note at both bases and harsh vesicular breath sounds at all areas; there were no accompaniments. On this occasion the temperature fell to normal by crisis four days later. On examination of the sputum no tubercle bacilli were found. The predominant organisms were Gram-positive lanceolate diplococci, morphologically resembling pneumococci; no streptococcal chains were seen. On X ray examination of the chest on May 21, 1935, dulness was found extending outwards from and downwards from the hilar region, with increase of lung markings extending upwards, suggestive of unresolved pneumonia. On June 3, 1935, three injections of albumose-free tuberculin were given subcutaneously at intervals of two or three days; no reaction occurred.

Dr. Griffiths said that the absence of reaction to the injections of tuberculin excluded tuberculosis; other investigations also failed to reveal the cause of the recurrence. It was difficult to know whether to call the second attack a relapse (*pneumonie à rechute*) or a recurrence (*pneumonie à récidive*); the length of the period of well-being suggested the latter. The boy was now very well and bright.

Arsenical Poisoning.

Dr. Griffiths also showed a patient who was suffering from arsenical poisoning. This case will be reported in full in a subsequent issue.

Splenic Anæmia.

DR. F. GUY GRIFFITHS and DR. V. M. COPPLESON showed a woman, aged twenty-six years, single, a dressmaker, who had been admitted to hospital on April 10, 1934, complaining of pain in the right side of the abdomen of ten days' duration and of pain in the left side of the abdomen. The pain on the left side had been first of all burning and then dragging for fourteen days. The history given by the patient was that she had been well until eight years ago, when she began to have attacks of vomiting. She was sent to hospital and remained there for three months. On her discharge from hospital she was much improved in health. Since this time, however, she had been pale and her pallor had been increasing. She then began to feel listless, was rapidly fatigued and had dyspnœa on slight exertion. Palpitation and œdema of the feet were present, but no giddiness or fainting attacks occurred. No cough or sputum was present. The patient was subject to frequent colds. She had occasional attacks of vomiting. Her appetite was poor and her bowels were constipated. She had no symptoms referable to the urinary system. At the time of her admission to hospital she had been losing weight for several months. She had never been jaundiced.

At the time of her admission to hospital examination revealed a very pale young woman with pale conjunctivæ and bluish sclerotics. No petechial hæmorrhages were present in the skin. Some glands were palpable in both inguinal regions. No glandular enlargement was found in the neck or in the axillæ. The spleen was greatly enlarged; it reached almost to the mid-line and was hard and tender. The pulse was regular; its tension was not increased and the vessel wall was impalpable. The heart was normal. Examination of the respiratory system revealed no abnormality. The pupils were equal and reacted to light and accommodation. Knee jerks and biceps jerks were present. There was no sensory or motor disturbance.

Inquiry into the family history revealed that the patient's brother suffered from splenic anæmia ten years ago. His spleen was removed and in a year he had grown well and strong.

On April 13, 1935, the Casoni test gave an immediate and delayed negative response. The Van den Bergh test gave a direct negative and an indirect positive response. Neither the Wassermann test nor the Kahn flocculation test yielded a reaction. Graham's test revealed a collection of gall-stones in a somewhat poorly functioning gall-bladder. The blood sedimentation rate was ten. A test was carried out to determine the fragility of the red blood cells. Incomplete hæmolytis occurred in 0·45% and complete hæmolysis in 0·40% saline solution; in the control incomplete hæmolysis occurred in 0·40% and complete hæmolysis in 0·35% saline solution.

On April 15, 1935, a blood count yielded the following information:

Erythrocytes, per cubic millimetre ..	2,770,000
Hæmoglobin value	49%
Colour index	0·89
Leucocytes, per cubic millimetre	4,800
Neutrophile cells	59%
Lymphocytes	35%
Monocytes	1%
Eosinophile cells	3%
Basophile cells	2%

Anisocytosis, poikilocytosis and polychromasia were present.

On June 5, 1935, a further blood count yielded the following information:

Erythrocytes, per cubic millimetre ..	2,950,000
Hæmoglobin value	53%
Colour index	0·9
Leucocytes, per cubic millimetre	6,000

Anisocytosis and slight polychromasia were present. The leucocytes appeared normal.

On June 12, 1935, Dr. Coppleson removed the spleen and the gall-bladder, in which five stones were present. The spleen weighed 695 grammes. The patient was doing well.

Diabetes Mellitus: Coronary Occlusion.

DR. W. W. INGRAM showed a female patient, aged twenty-five years, a diabetic, who had been for several years stabilized on diet and insulin. Her urine had remained sugar-free; her weight had been satisfactory and her blood sugar estimations had been satisfactory.

Ten weeks prior to seeking advice she missed a period and since then had had complete amenorrhœa. During this period she had also lost 12·6 kilograms (two stone) in weight, with progressive loss of energy, and for three weeks had had giddy turns with dyspnœa. She had not at any time had any pain or cough when she was admitted as an in-patient for investigation.

Examination of her chest revealed no abnormality, and a skiagram of her chest was normal. Her basal metabolic rate was +12%. Her pulse was regular and forceful. Her systolic blood pressure was 120 and her diastolic pressure 90 millimetres of mercury, the heart sounds were normal, and the heart was normal in size. Nothing abnormal was found in her nervous or alimentary systems. A blood count revealed a total red cell count of 5,500,000 per cubic millimetre. The hæmoglobin value was 98% and the total leucocyte count was 7,700 per cubic millimetre. An electrocardiogram taken on May 20, 1935, showed a typical coronary occlusion, and one taken subsequently before her

discharge showed an abnormal *T* wave in the fourth lead, with probable coronary sclerosis.

With rest in bed all the patient's symptoms had disappeared and she was discharged quite well. The patient never at any time had pain in the chest or abdomen, and the presenting signs were loss of weight and breathlessness.

Coronary Occlusion.

Dr. Ingram also showed a male patient, aged forty-two years, a farmer of South Africa, who had had no previous illnesses and who complained of intense substernal pain radiating down the inner aspect of the left arm and into the epigastrium. The pain had come on suddenly and he vomited and rolled about in agony. When seen, he was very pale; he kept absolutely still and was afraid to move. His pulse was regular; the cardiac apex beat was in the fifth interspace and 8·75 centimetres (three and a half inches) out; there was no thrill. The heart sounds were poor in quality, but normal in rhythm. His systolic blood pressure on admission was 111 millimetres and the diastolic pressure 90 millimetres of mercury, but later the figures were 90 and 65. The Wassermann test gave no reaction and his urine showed a trace of sugar on the second day. He had no rise of temperature and no leucocytosis.

With rest in bed his systolic and diastolic blood pressure rose to 120 and 90 millimetres of mercury respectively, but when he was allowed to sit up it again fell to 90 and 64 millimetres, and every time an attempt was made to get the patient up, even after two months in bed, there was a sudden fall in blood pressure, necessitating further rest in bed, and no further attempt had been made to get him up.

An electrocardiogram on April 17, 1935, showed the typical changes due to recent acute coronary artery occlusion. On May 17, 1935, it showed the typical changes of late stage following an acute coronary artery occlusion, and on June 4, 1935, further improvement was noted by the *T* wave in Lead I, which was now only slightly indented.

Addison's Disease.

Dr. Ingram's next patient was a woman, aged thirty-two years, of Lithuanian extraction and of Semitic parentage. She was quite well until twelve months ago, when she felt "run down". She kept at work, however, and in November, 1934, she had an acute attack of indigestion. Following this she became progressively more fatigued, until at Christmas time she became so weak that she could scarcely walk across the room. Since November she had lost 7·6 kilograms (one stone three pounds) in weight; she had had anorexia and insomnia. Also she noticed her skin becoming more pigmented. She had no cough, sputum or pain in the chest, no night sweats or hæmoptysis. Occasionally she was short of breath and had slight œdema of the feet. She slept well and dreamlessly. She had slight leucorrhœa and her menstrual loss had been more scanty since the onset of fatigue. She had previously had diphtheria and rheumatic fever.

The pulse was small in amplitude and volume. The heart was of normal size. There was a faint mitral systolic thrill and a reduplicated mitral first sound. The systolic blood pressure was 88 and the diastolic pressure was 60 millimetres of mercury. There was marked pigmentation of the face, flexor surfaces of the knees and elbows, becoming progressively more pronounced. The mucous membrane of the mouth and tongue was deeply pigmented. No abnormality was found in the respiratory, central nervous or genito-urinary systems.

A blood count revealed 4,850,000 red cells per cubic millimetre, a hæmoglobin value of 86%, a total leucocyte count of 6,650 per cubic millimetre, with 37% polymorphonuclear cells and 57% lymphocytes.

The basal metabolic rate was −12%, and the blood sedimentation rate was 19. The blood sugar content one hour after a meal was 0·09%. X ray examination of the chest revealed chronic bronchitic changes only. An X ray examination of the suprarenal area revealed scoliosis of the lumbar region, but no calcification of the suprarenals was detected.

The patient had had attacks characterized by vomiting and nausea, anorexia, severe headache, drop in both systolic and diastolic blood pressures, associated with extreme lassitude merging on collapse. The attacks had occurred at intervals of approximately one week since the patient's admission to hospital.

Treatment had included the intramuscular injection of the suprarenal cortical extract "Eschatin". The patient had been given at first two cubic centimetres every other day and the dose had been increased to three cubic centimetres per day. Sodium chloride was given by mouth, both with food and in capsules; cotton seed oil was given in capsules three times a day, and a high fat diet was ordered. The patient was also given normal saline solution and glucose *per rectum* during the remissions.

Since her admission to hospital the patient's pigmentation had become less marked, but there had been no striking improvement in her general condition.

Thrombo-Anglitis Obliterans.

Dr. Ingram showed a male patient, aged forty-five years, who came from the Spanish border of France. The patient had been quite well until 1915, when he was hit on the right knee by a piece of shell at the War. After his discharge from the army in 1916 he had pain in both legs. He spent some time in Broughton Hall for "trembling". He managed to get about for some years, but in 1925 he developed an ingrowing toe nail. This would not heal, and in 1927 his great toe was amputated. Later in that year he developed an ulcer on the right foot and the left knee was kicked by a horse. Both legs became progressively worse and were both amputated. In January, 1934, his hands felt cold and went blue, perionychia appeared and the fingers became gangrenous and the index and middle fingers were amputated from the right hand.

The patient on admission to hospital complained that there was blueness, soreness and coldness of the remaining fingers of the right hand extending over a period of fifteen months. During this time there had been stiffness and decreased ability to move the fingers. He smoked one ounce of tobacco a week and was a very moderate drinker. There was no family history of this complaint.

The pulse in the right radial artery was not palpable, but it was palpable in the left radial and both brachial arteries. The heart was not enlarged to percussion, but the heart sounds were hardly audible, and the systolic blood pressure in the right arm was 90 and the diastolic pressure was 58 millimetres of mercury; in the left arm the pressures were 90 and 68 millimetres. No abnormal signs were found in the respiratory or central nervous system and the Wassermann test gave no reaction.

An X ray examination of the chest revealed no abnormality and X ray examination of the limbs showed no evidence of calcified vessels. His total red blood cells were 5,500,000 per cubic millimetre, with a hæmoglobin value of 104%, and the total leucocytes were 11,300 per cubic millimetre. The colloidal gold curve in the central nervous system was 110011110000. The blood phosphorus content was 2·4 milligrammes *per centum* and the blood calcium was 13·28 milligrammes. The result of a urea concentration test was 0·1%; one hour after the draught the figure was 0·08%, and two hours after the draught 0·06%. The ring and little fingers of the right hand showed blueness, and the little finger had incipient perionychia.

The ulnar nerve was crushed and the ulnar artery was decorticulated. The pain in the third finger continued and was relieved only by morphine. It was amputated and intravenous normal saline solution was given. Microscopic examination of a section of the arteries showed an area of connective tissue around nerves and artery.

Various forms of treatment had been tried, including intravenous therapy, but nothing had been found which had any effect on the progress of the disease.

Lipiodol in Pulmonary Disease.

DR. COTTER HARVEY showed a series of skiagrams illustrating the value of lipiodol in pulmonary disease, with special reference to bronchiectasis.

Various types of bronchiectasis were demonstrated by the bronchograms, the cylindrical, fusiform, saccular, moniliform, dry hæmorrhagic and atelectatic, all being illustrated.

Cases were also demonstrated wherein provisional radiological diagnoses had been refuted by the aid of lipiodol, and in which an accurate estimation of the nature and extent of the pathological process had been obtained, information which the plain skiagram had failed to reveal.

Tendon Transplantation for Ocular Paralysis.

DR. E. TEMPLE SMITH showed a man, aged fifty-nine years, on whom, six weeks previously, he had performed the operation of tendon transplantation for paralysis of the sixth nerve. The patient had had paralysis of the left abducens for twenty months. He had had constant diplopia when looking ahead, and in order to carry on his occupation (of hairdressing) he had to occlude one eye or to work and to move with the head well over to the right in a crab-like manner. His disability was considerable.

Slips from the superior and the inferior recti muscles were grafted into the insertion of the paralysed muscle and this was combined with tenotomy of the medial rectus of the same eye.

Dr. Temple Smith pointed out that the patient had now no diplopia in any position except extreme lævoduction, he was perfectly comfortable walking and working, and there was about 45° of movement to the left.

Dependent Drainage of Appendiceal Abscess.

DR. L. S. LOEWENTHAL showed two children on each of whom dependent drainage of appendiceal abscess had been instituted by incision *per rectum*. He referred to the anxiety of the conscientious surgeon in dealing with neglected cases of acute appendicitis, and to the risks attendant on drainage of appendiceal abscess by exploratory laparotomy. The method described was, in his opinion, practically devoid of risk and of unpleasant sequelæ. He considered it to be without doubt the method of election in children and men, and even to be preferred to vaginal cœliotomy in adult women.

The technique, apparently not referred to in standard text books, was simple. With the patient in the lithotomy position, the forefinger of the left hand was passed through the *sphincter ani externus*, which was usually relaxed in these cases. A long aspirating needle was passed anterior to the finger, through the rectal wall, into the swelling, generally presenting at a distance of about five centimetres from the anus. If pus was obtained before removal of the needle, a knife was passed along the track into the swelling, the incision thus made being widened by Hilton's method. A large rubber drainage tube was then inserted and stitched to the buttock. The patient was nursed in Fowler's position and the tube was left *in situ* for seven to ten days. No difficulty had been experienced with the bowels following the above procedures.

The first patient, a girl aged twelve years, had been admitted to hospital seven months previously with a history of abdominal pain and vomiting commencing two weeks prior to admission. She had been investigated by the urologist, who reported a large cystic swelling in the pelvis behind the bladder. A diagnosis of pelvic appendiceal abscess was made and the mass was opened *per rectum*, about fifteen ounces of foul-smelling pus being evacuated. Six weeks after her discharge from hospital the child had returned with a twenty-four hour history of abdomonal pain, vomiting and pyrexia. Under general anæsthesia, rectal examination revealed a mass high up on the right wall of the pelvis. On abdominal exploration this was found to be an abscess; it was drained through the incision, no attempt being made to remove the appendix. Three months later appendicectomy had been performed, the appendix being found adherent in the pelvis. The child was now perfectly well.

Dr. Loewenthal's second patient was a boy, aged thirteen years, who had been admitted to hospital in January with a history of abdominal pain and vomiting for seven days. The lower part of the abdomen was rigid and exquisitely tender, and no mass could be felt. The diagnosis of general peritonitis following a ruptured appendix was verified at operation. The appendix was not removed, a drainage tube being inserted through the incision in the right iliac quadrant. Four weeks later a pelvic abscess was drained *per rectum*, nearly one pint of foul pus being evacuated. One month prior to the meeting appendicectomy was performed through the original incision, in which there was a small herniation at the site of the tube sinus; the appendix was found firmly adherent in the pelvis. Convalescence had been uneventful and the patient appeared quite well.

Nephrectomy for Hypernephroma.

DR. COLIN EDWARDS showed a male patient, aged forty-seven years, married, a bicycle repairer, who was admitted to hospital in March, 1932. The patient gave a history that two months before admission he was seen by his own doctor for pain in the left iliac fossa. At this time a tumour was felt in the left renal region, but the patient declined investigation. The day before his admission to hospital he passed urine which appeared to be mainly blood. The only pain was a slight "stitch" in the left side of the abdomen.

On examination the patient was very obese. A large mass was palpable in the left renal region. It was immobile and was not tender. Apart from emphysema, no other abnormality was found. Microscopical examination of the urine revealed blood cells, blood and granular casts. On culture a heavy growth of Gram-negative coliform organisms was obtained. The blood urea was 25·9 milligrammes per 100 cubic centimetres of blood. X ray examination revealed no abnormality. Intravenous pyelography showed that the right kidney was normal in shape and position, but larger than normal. The function was good. The left side showed distortion due to a tumour of the upper pole. The opaque substance appeared later than on the right side.

Nephrectomy was performed. The kidney and tumour were 22·5 centimetres (nine inches) in length. The upper portion was 17·5 centimetres (seven inches) wide, but the lower part appeared to be normal kidney substance. The tumour was particularly adherent to the posterior abdominal wall and to the peritoneum anteriorly and superiorly. The para-aortic glands were necrotic and were removed by hand in large soft pieces, but the renal vein was not encroached upon. The tumour was ruptured during removal and the wound became infected.

Since the operation the patient had gained in weight and had carried on a fairly arduous occupation. Frequently he might be seen pedalling a bicycle up Taverner's Hill. There was an incisional hernia at the site of the drainage tube, which did not greatly inconvenience him.

Dr. Walton Smith had reported that the growth showed some departure from the usual hypernephroma: (*a*) macroscopically it was not well marked off from the kidney tissue; (*b*) microscopically there were some cells of epithelioid type in rather scattered arrangement, but the main mass of the growth was made up of cells of the spindle cell type and so resembling more a sarcoma than a hypernephroma, but these cells did occur in the latter. There were large necrotic areas and much hæmorrhage, also areas of fibrous overgrowth, so that, in Dr. Walton Smith's opinion, it might be classed as an atypical hypernephroma.

Dr. Edwards also showed a male patient, aged fifty-five years, married, a shearer, who was admitted to hospital in March, 1934.

According to the history the patient had complained of pain in the right loin for two months and of hæmaturia for five weeks. There was a firm immobile tumour of the right hypochondrium, also palpable in the right costovertebral angle. Culture of urine revealed no abnormality. On X ray examination the right psoas margin of the kidney was obscured. Intravenous pyelography revealed a normal outline of the left kidney with good function; no

excretion occurred on the right side. On cystoscopic examination the bladder was normal. A good blue colour was obtained from the left side after intravenous administration of indigo-carmine. Instrumental pyelography revealed slight hydronephrosis with gross deformity of the pelvis, due to a tumour of the upper pole of the right kidney.

Nephrectomy was performed. The kidney and tumour measured 30 by 20 centimetres (12 by 8 inches). There were many adventitious veins from the renal capsule and dense adhesions to the peritoneum anteriorly.

Dr. Walton Smith had reported that the specimen was a large growth with a small portion of kidney tissue at the lower pole. It showed numerous cysts filled with gelatinous material, large blood spaces, hæmorrhages and fibrous bands. The tumour was one which was usually classed as hypernephroma, but in the structure there were numerous lumina (microscopically these closely resembled thyreoid gland) and, according to Ewing, these tumours should be classed as adeno-carcinoma with clear cells arising from the kidney itself and not from adrenal rests. In some of the cysts there were papillary ingrowths.

Dr. Edwards said that the patient was very well and had gained in weight. He had not been able to resume arduous work owing to weakness of the right abdominal wall, probably due to nerve injury during removal of the tumour. He was able to work in the garden.

Incontinence of Urine following Perineal Prostatectomy.

Dr. Edwards's last patient was a man, aged sixty-four years, married, an engine-driver, who had been admitted to Lidcombe State Hospital in February, 1935. He had complained of incontinence of urine and scalding on micturition since perineal prostatectomy two and a half years previously. At another hospital the following operations had been performed: appendicectomy, colostomy, partial colectomy, cholecystectomy, suprapubic cystostomy and perineal prostatectomy. He also suffered from "colitis" with frequent watery stools.

On examination the abdomen was much scarred, but otherwise normal. Rectal palpation gave the sensation of a complete removal of the prostate. Cysto-urethroscopy showed a normal bladder wall, but there was an irregularity of the internal urinary meatus suggesting the recurrence of hyperplasia in a nodule of prostatic tissue and damage to the internal sphincter muscle. The external sphincter moved slightly with voluntary effort, but the posterior portion was immobile. There was one ounce of residual urine. It contained pus and Gram-negative organisms. The results of renal function tests were good.

H. H. Young's operation was performed: (*a*) Transvesical exposure of the bladder neck and the submucous muscular layer of the internal sphincter. Fibrous tissue was dissected away, the muscle was reunited posteriorly and the whole was covered with mucosa. A suprapubic drain was used. (*b*) Perineal exposure of the external sphincter. Fibrous tissue was dissected away and the muscle was tightened by suturing over a small sound.

Dr. Edwards pointed out that the patient was well, apart from constipation. He was completely continent and did not have to void urine at night.

Skiagrams.

DR. H. R. SEAR demonstrated reproductions of skiagrams showing various types of *leontiasis ossea*, both periostitic and osteitic, Paget's disease of the skull in different stages and Paget's disease of the mandible, also fibrocystic disease of the mandible and skull.

(To be continued.)

NOMINATIONS AND ELECTIONS.

THE undermentioned has been elected a member of the New South Wales Branch of the British Medical Association:

Uren, Harry Keith, M.B., B.S., 1933 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Correspondence.

X RAY TREATMENT OF ACNE.

SIR: With reference to Dr. Belisario's paper on the treatment of *acne vulgaris*, I should like to sound a note of warning.

No one can deny that X rays are the most rapidly effective and dramatic of all remedies advocated for this disease. But acne is a disease which carries no threat to the patient's life and little to his health. Therefore, risks that might be justifiable in a more serious disease are not warranted in acne.

There are many grades of severity in *acne vulgaris*, and to advocate the use of X rays in all cases shows, to my mind, too great enthusiasm or a faulty sense of proportion. The majority of patients, especially girls who care enough about the disfigurement to perform the treatment regularly and efficiently, can be cleared of acne and can be kept clear without ever using X rays and without any risk at all, and at a much smaller cost than that implied in the delivery of the long series of X ray treatments advocated by Dr. Belisario.

The method described in my paper published in THE MEDICAL JOURNAL OF AUSTRALIA, July 3, 1926, still remains my sheet anchor in the treatment of *acne vulgaris*, and many practitioners in the city and country who have adopted it as a routine measure continue to assure me that it is one of the most efficient measures in their therapeutic armamentarium.

It is quite true that in bad cases a preliminary course of three or four quarter doses, or a single application of a three-quarter dose of X rays of the quality advocated by Dr. Belisario, makes an almost magical improvement and gives the patient heart to continue the manipulative treatment. But to continue the X radiation until no post-radiation measures are necessary is, to my mind, quite unjustifiable.

My reasons for this opinion are as follows: It is admitted that the action of X rays is not directly antibacterial. The reason for the success which follows their application is that they provide a very depressant effect upon the secreting membrane of the sebaceous glands. This results in a great diminution in the amount of sebaceous secretion which provides a culture medium for the acne bacillus. In consequence of diminution of food supply the acne bacilli are to a large extent starved out. But the sebaceous glands, especially during adolescence, soon recover their activity and conditions favourable to a recrudescence are again provided, unless measures for removal of the excess secretion are adopted.

If X rays are continued until the sebaceous glands do not resume their rate of secretion normal for the individual patient at this period of life, it can only be because permanent lowering of the activity of the secreting cells has been produced by the X rays. It is obvious that such a permanent effect is brought about by the determination of a degree of atrophy in the secreting cells.

But the sebaceous glands lie deeper than the basal layer of the epidermis proper, which by an immutable physical law receives and absorbs a larger amount of X radiation than any deeper structures and must be damaged to an extent not less than that suffered by the secreting membrane of the sebaceous glands.

This damage, it is true, is only mild and may be accompanied by no visible signs for a considerable period of years. But that the danger is a real one must be admitted by all. Quite recently a paper published by Stephan Epstein, "*Die Bedeutung der Überschneidungen für das Zustandekommen von Röntgenspätschäden in der Oberflächentherapie*" (*Archiv für Dermatologie und Syphilis*, Volume CLXVIII, Number 3) gives twelve instances of damage appearing long after the cessation of X ray treatment delivered in fractional doses similar in total and even longer in time of distribution than that advocated by Dr. Belisario.

It is to be noted that nearly all the support for the use of X rays exclusively or mainly in the treatment of acne comes from America where the statute of limitations provides for a period of only three years. In other countries, where the statute provides a six-year period, X ray damage would appear to occur more frequently, or at least frequently enough to damp the enthusiasm for prolonged X ray treatment of *acne vulgaris*.

If a woman at thirty years of age finds herself afflicted with a wrinkled atrophic senile skin as a result of having been treated at seventeen with a long series of X rays for acne, she will be not less resentful because she finds that she has no legal redress.

Speaking for myself, if my daughter had acne, I cannot imagine any argument that would persuade me to agree to a treatment with X rays up to the total dose suggested by Dr. Belisario, or even to half that amount, especially when other efficient methods are available, the use of which implies no risk at all.

Yours, etc.,
E. H. MOLESWORTH.

235, Macquarie Street,
Sydney,
November 13, 1935.

AN INTERMEDIATE OPHTHALMIC SERVICE.

SIR: I should like to comment on the letter by Dr. Edward L. Gault, appearing in the journal of November 9, on the intermediate ophthalmic service started in Sydney.

The Medical Eye Service of New South Wales was not established on the supposition that the private fees of oculists were high or that they required to be reduced, but rather upon the knowledge that they were beyond the means of a section of the community.

As matters stand today, prospective patients can consult an ophthalmologist privately, or, if they are unable to afford to pay his fees, they may consult him at a public hospital. There are many people who do not wish to go to hospital, where they pay the hospital fee and lose one or more days' wages; while they cannot afford private fees, they are definitely not indigent poor. Hitherto no provision has been made for this not inconsiderable number, and to remedy this the Medical Eye Service was instituted.

Dr. Gault states that "the magnitude of this social experiment has not received sufficient consideration". While this may be argued, it can be safely assumed that the question was very closely considered when the National Ophthalmic Treatment Board was established in England; and whether this Board is an experiment or not, some change was needed.

The question is, as Dr. Gault states, the safeguarding of the patient and of the profession, and he goes on to suggest that with the lowered fee the work may be proportionately less thorough. I would ask does his suggestion apply to the honorary work done at public hospitals.

The income limit imposed at the Medical Eye Service of New South Wales is five pounds per week, but it is found that the majority of patients seeking attention there have incomes well below that limit. It must be admitted that some of the patients seen have consulted an ophthalmologist privately, or would have done so. The majority have not consulted any doctor, and of those who have done so, the greater proportion have seen the doctor at hospital.

The contention that the Medical Eye Service transgresses a cardinal principle of medical practice, namely, the preservation, as far as possible, of the relationship between doctor and patient and freedom of choice by the patient of his medical adviser, cannot be upheld. The patient has this choice at the clinic, and the work done there by the surgeon is in effect part of his private practice, and consultations are carried out precisely as at his rooms.

Any ophthalmologist who is a member of the British Medical Association and of the Ophthalmological Society of New South Wales may take, and can demand to take, a session, afternoon or evening, at the Service, which is most emphatically not a "close corporation". And similarly, if he does not wish to work there, he can be a member and have equal voice in the conduct of the service, even to voting it out of existence.

The Medical Eye Service is not different from any clinic, except that it is controlled by medical men. Its success depends on the efficiency of the work done and on the demand for the service. Its conduct and policy are in the hands of members of the profession, and no practising specialist is debarred from working there or from voicing his opinion and influencing the policy or management.

Should any of "the other objectionable features" not mentioned by Dr. Gault arise, there is no reason why they should not be corrected, or, as I have mentioned above, why the whole service should not be voted out of existence.

In conclusion, I think there is a definite need for such a service, and the results of its twelve months' trial only serve to strengthen that opinion.

Yours, etc.,
WILLIAM M. C. MACDONALD.

235, Macquarie Street,
Sydney,
November 15, 1935.

TREATMENT BY OLIVE OIL EMULSION.

SIR: It was interesting to read in THE MEDICAL JOURNAL OF AUSTRALIA, of November 9 of a case in which dramatic improvement had occurred following intravenous injection of olive oil emulsion.

So far as I am aware this is the first case of the kind reported in any medical journal, and the writer very properly points out that when a series of cases has been published comments will then have some statistical value. It is unfortunate, in the absence of such necessary statistical data, that this method of treatment has received such wide publicity in the lay Press both here and in England. This fact and the lack of adequate data to support the claims made on its behalf were subjects of scathing editorial comment in *The British Medical Journal* of March 30 this year.

From the scientific point of view several aspects of the subject call for comment, but I shall limit my remarks to consideration of only two. First, the adsorption of toxins may be a plausible hypothesis, but lacks adequate proof. It was first put forward as a rationale for injection of various colloidal preparations more than twenty years ago. Subsequent experience has provided little, if any, evidence of their value. One finds very little reason to believe that toxins, actual or hypothetical, are selectively adsorbed by an emulsion of olive oil. Secondly, one would remind readers of some, perhaps partly forgotten, facts regarding the physiology of fat absorption from the intestine. Fats, including olive oil, are split into glycerol and soluble fatty acids, which pass through the intestinal villi into the lacteals, where they recombine as fats and pass up the thoracic duct in the form of a fine emulsion, entering the circulation at a point not far distant from the usual site of an intravenous injection.

Assuming for the moment that olive oil emulsion may be of benefit, the oral and intravenous routes of administration differ only in three respects: (i) a distance of about eighteen inches between the points of entry into the venous circulation; (ii) a period of three or four hours required for the digestion and absorption of the oil when administered orally; (iii) the psychological effect, upon the patient and relatives, of an intravenous injection of a substance hailed by the lay Press as the latest marvel of medical science may possibly be commercially advantageous.

Yours, etc.,
H. WHITRIDGE DAVIES,
Professor of Physiology.

Department of Physiology,
University of Sydney,
November 15, 1935.

Books Received.

CLINICAL PARASITOLOGY AND TROPICAL MEDICINE, by D. de Rivas, B.Sc.Biol., M.S., M.D., Ph.D., in collaboration with C. T. de Rivas, B.A., M.D.; 1935. Philadelphia: Lea and Febiger. Royal 8vo., pp. 367, with illustrations. Price: $5.00 net.

ANATOMY, DESCRIPTIVE AND APPLIED, by H. Gray, F.R.S., F.R.C.S.; Twenty-Sixth Edition, edited by T. B. Johnston, M.B., Ch.B.; 1935. London: Longmans, Green and Company. Royal 8vo., pp 1548, with 1,323 illustrations, of which 611 are coloured. Price: 42s. net.

UROLOGY IN GENERAL PRACTICE, by A. E. Roche, M.A., M.D., M.Ch., F.R.C.S.; 1935. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 366, with illustrations. Price: 17s. 6d. net.

THE SPLEEN AND RESISTANCE, by D. Perla, M.D., and J. Marmorston, M.D., with a foreword by D. Marine, M.D.; 1935. London: Baillière, Tindall and Cox. Royal 8vo., pp. 181. Price: 9s. net.

BACTERIA IN RELATION TO THE MILK SUPPLY: A PRACTICAL GUIDE FOR THE COMMERCIAL BACTERIOLOGIST, by C. H. Chalmers, B.Sc., N.D.A., with a foreword by A. T. R. Mattick, B.Sc., Ph.D.; 1935. London: Edward Arnold and Company. Crown 8vo., pp. 204, with illustrations. Price: 6s. net.

MANUAL OF HUMAN PHYSIOLOGY, by Sir Leonard Hill, M.B., LL.D., F.R.S., Hon. A.R.I.A.; Fourth Edition; 1935. London: Edward Arnold and Company. Crown 8vo., pp. 482, with illustrations. Price: 6s. 6d. net.

LOCALIZED RAREFYING CONDITIONS OF BONE AS EXEMPLIFIED BY LEGG-PERTHES' DISEASE, OSGOOD-SCHLATTER'S DISEASE, KUMMELL'S DISEASE AND RELATED CONDITIONS, by E. S. J. King, M.D., D.Sc., M.S., F.R.C.S., F.R.A.C.S.; 1935. London: Edward Arnold and Company. Royal 8vo., pp. 412, with illustrations. Price: 35s net.

Diary for the Month.

Nov. 23.—Victorian Branch, B.M.A.: Country Branch Meeting (Horsham).
Nov. 26.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 27.—Victorian Branch, B.M.A.: Council.
Nov. 28.—South Australian Branch, B.M.A.: Branch.
Nov. 28.—New South Wales Branch, B.M.A.: Branch.
Dec. 2.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Dec. 3.—Tasmanian Branch, B.M.A.: Council.
Dec. 3.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Dec. 4.—Western Australian Branch, B.M.A.: Council.
Dec. 4.—Victorian Branch, B.M.A.: Annual Meeting.
Dec. 5.—South Australian Branch, B.M.A.: Council.
Dec. 10.—Tasmanian Branch, B.M.A.: Branch.
Dec. 10.—New South Wales Branch, B.M.A.: Ethics Committee.
Dec. 12.—New South Wales Branch, B.M.A.: Branch.
Dec. 13.—Queensland Branch, B.M.A.: Annual Meeting.
Dec. 17.—Tasmanian Branch, B.M.A.: Council.
Dec. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Dec. 20.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii, xix. and xx.

LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Pathologist and Biochemist.

PUBLIC SERVICE BOARD, ADELAIDE, SOUTH AUSTRALIA: Director of the Government Laboratory of Bacteriology and Pathology.

PRINCE HENRY'S HOSPITAL, MELBOURNE, VICTORIA: Resident Medical Officer.

QUEEN'S MEMORIAL INFECTIOUS DISEASES HOSPITAL, FAIRFIELD, VICTORIA: Senior Assistant Resident Medical Officer.

SAINT VINCENT'S HOSPITAL, MELBOURNE, VICTORIA: Medical Superintendent.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chiliagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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